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DIFFERENTIAL DIAGNOSIS

Growth and development

1. Short stature.

Definition:

Height below the 5th percentile for age and sex

1. Normal variants (90%)

	Familial (genetic)	Constitutional delay of growth and puberty
Parents	Short	Normal height
Growth velocity	Normal (short since birth)	Normal length at birth → short within 2 years → transient decelerated growth
Bone age	Normal	Delayed
Puberty	Normal	Delayed
Adult eight	Short adult height	Normal adult height
Treatment	GH may be useful	Reassurance

- 2. Pathological (10%)
- A. Endocrinal causes: (bone age markedly delayed)

a. Growth hormone deficiency

- Panhypopituitarism
- Isolated GH deficiency
- 2ry to craniopharyngeoma: (visual field assessment and CT brain are important to exclude it.
- Laron syndrome: IGF1 deficiency.
- b. Hypothyroidism: (congenital/ autoimmune thyroiditis)
- c. Hypoparathyroidism
- d. Adrenal gland:
 - Adrenal insufficiency
 - Cushing syndrome
 - Corticosteroid therapy: a prolonged high dose (equivalent to 5mg prednisone / day or more leads to short stature (Risk is reduced by alternate day therapy)
- B. Genetic and chromosomal disorders:
 - a. Down syndrome
 - b. Turner syndrome
 - c. Silver Russel syndrome
 - d. Skeletal dysplasia e.g. achondroplasia
 - e. Mucopolysaccharidosis
- C. Severe IUGR:

Some may show persistently restricted growth (GH therapy)

- D. Nutritional starvation:
 - Nutritional starvation diminishes synthesis of growth factors
 - Weight is more involved than the height
- E. Social short stature:

Psychological deprivation: Disturbed child mother or family relation → reduce growth hormone release

- F. Severe systemic diseases in infancy and childhood:
 - Chronic diseases (renal failure LCF malabsorption hemolytic anemia)
 - Chronic infections: Tuberculosis bilhariziasis

2. Macrocephaly.

Definition:

Head circumference > 95th percentile for age & sex

Etiology:

- A. Cranial causes:
 - Familial large head.
 - Chronic hemolytic anemia.
 - Rickets
 - Achondroplasia
- B. Intracranial causes (causes of increased intracranial tension)
 - Hydrocephalus
 - Subdural hematoma
 - Subdural effusion
 - Brain tumors
 - Neurofibromatosis
 - Cerebral gigantism (Sotos syndrome)
 - CNS storage disorders, e.g. mucopolysaccharidosis (Ruler's syndrome)

3. Microcephaly.

- **→** *Etiology:*
 - 3. True microcephaly:
 - A. Primary (genetic):
 - a. Familial (autosomal recessive).
 - b. Autosomal dominant.
 - c. Syndromes: Down (trisomy 21), Edwards (trisomy 18), Cri du chat (deletion of tip of short arm of chromosome no. 5).
 - B. Secondary (non genetic or acquired):

Destructive process affecting the brain during fetal & early infancy.

- i. <u>Prenatal causes:</u>
 - a. Congenital infection: as rubella & cytomegalovirus (TORCH).
 - b. Irradiation.
 - c. Toxins.
 - d. Drugs: fetal alcohol syndrome, fetal hydantoin syndrome.

ii. Natal causes:

Hypoxic ischemic injury.

iii. Postnatal causes:

- a. Kernicterus.
- b. CNS infection: meningitis-encephalitis.
- c. Intracranial hemorrhage.
- 4. Craniostenosis (craniosynostosis):

Premature closure of skull sutures.

When generalized (multiple sutures), it leads to microcephaly with motor & mental retardation.

- A. Isolated congenital defect.
- B. Genetic syndromes: as Crouzon syndrome (+ exophthalmos).

4. Tall stature.

Etiology:

Proportionate tall stature	Disproportionate tall stature	
Familial tall stature	Marfan syndrome	
Exogenous obesity	Homocystinuria	
Precocious puberty	Klinefelter	
GH excess (Gigangtism)	Cerebral gigantism (Sotos syndrome)	

5. DD of delayed walking.

- 1. Nutritional:
 - PEM
 - Rickets
- 2. Chromosomal
 - Down
 - Prader Willi
- 3. Endocrinal:
 - Cretinism
- 4. Neurological:
 - CP
 - Hydrocephalus
 - Werdnig Hoffman
- 5. Skeletal:
 - Osteogenesis imperfecta
- 6. Environmental:
 - Lack of training
- 7. Familial / normal variant
- 8. Sensory:
 - Interference with perception of movement

Nutrition

- 1. DD of kwashiorkor.
- 1. From other causes of generalized edema (cardiac, hepatic renal & allergic)
- 2. From napkin dermatitis & pellagra
- 3. From other causes of immunodeficiency
- 2. DD of failure to thrive (secondary marasmus).
- **→** Definition:

Hight or weight less than 3rd percentile for age

- **→** Causes:
 - 1. Inadequate intake:
 - a. Non-organic (nutritional):
 - i. Decreased food:
 - Causes of marasmus
 - Difficult child
 - Feeding disorders (لا يكبر)
 - ii. Psychological depression
 - Maternal depression
 - Low education
 - Child abuse
 - b. Organic (non nutritional):
 - CP
 - Cleft lip and palate
 - Esophageal stricture (corrosive)
 - Congenital pyloric stenosis (CHPS)
 - 2. Loss of nutrients:
 - Vomiting
 - Diarrhea
 - 5. Malabsorption:
 - Celiac and cystic fibrosis
 - 6. Poor utilization:
 - Down syndrome
 - IUGR and extreme prematurity
 - Inborn error of metabolism
 - System failure (liver)
 - 7. Increased requirements:
 - Thyrotoxicosis
 - Malignancy
 - Congenital heart disease
 - Chronic infections (TB,UTI)
 - DM

Infection

1. Short febrile illness.

- More common and serious
- Less than 7 days
- A) Focal infection:
 - 1. Simple Focal infections:
 - a. **Respiratory** (URTI is the most common)

Nasopharyngitis, otitis media, sinusitis, bronchitis

- b. **Digestive:** stomatitis, gastroenteritis
- c. <u>Urinary:</u> Urinary tract infection "cystitis"
- d. Cutaneous: cellulitis, abscess
 - Detailed examination and history can discover the focus
 - ENT examination is essential "otitis media is common"
- 2. Serious Focal infection:
 - a. <u>Meningitis:</u> disturbed consciousness, convulsions, meningeal irritation Increased intracranial tension "headache, projectile vomiting, blurred vision"
 - b. **Pneumonia:** Respiratory Distress, Rales, Bronchial breathing
 - c. Pyelonephritis: loin tenderness or swelling, turbid urine or hematuria
 - d. **Peritonitis:** abdominal distension and generalized tenderness
 - e. Osteomyelitis or arthritis: tenderness, swelling, limitation of movement

N.B.

Early focal infection "first 24 or 48 hours", the focus may not be evident (Re-examination after 24 or 48 hours reveals the focus in up to 40% of cases)

B) Simple fever "Non -specific fever"

Clinical diagnosis depends on:

- 1. Degree of fever
- 2. History: Appetite Activity Reaction to parents
- 3. Examination: \underline{A} ppearance \underline{A} lertness \underline{R} esponse to social stimuli
- i. Viremia:
 - <u>Fever</u>: low grade
 - History and examination: Normal
 - <u>Investigations</u>: not needed
 - Treatment:
 - Antipyretic
 - Re-examination after 24-48 h. a focus may be found
- ii. Bacteremia:
 - Fever: High grade
 - History and examination: sick
 - Investigations:
 - 1. CBC: leukocytosis >15000/mm3
 - 2. Band cell >10%

- 3. CRP: elevated to 20-30 mg/l
- 4. ESR >20 in 1^{st} hour

• Treatment:

- Oral broad-spectrum antibiotic (ampicillin or amoxicillin)
- Re-examination after 24-48 hours

iii. Septicemia:

See emergency.

2. Prolonged fever

- → Duration more than 10-14 days
- **→** *Etiology:*
 - A) Infection "most common"
 - 1. Bacterial:
 - > Systemic infection
 - a. Salmonellosis
 - b. Brucellosis
 - c. Tuberculosis
 - d. Listeriosis
 - > Hidden focal
 - a. Abdominal abcess
 - b. Endocarditis
 - c. Pyelonephritis
 - d. Osteomyelitis
 - 2. Viral:
 - a. Infectious mononucleosis
 - b. CMV
 - c. HIV
 - d. HCV
 - 3. Parasitic:
 - a. Malaria
 - b. Toxoplasmosis
 - c. Visceral larva migrans
 - B) Autoimmune
 - 1. Rheumatic fever
 - 2. Juvenile rheumatoid arthritis
 - 3. SLE
 - C) Malignancy
 - 1. leukemia
 - 2. lymphoma
 - 3. Neuroblastoma

3. Fever with purpuric rash.

- 1. Serious bacterial infection (20%):
 - a. Meningococcal septicemia is the most common.
 - b. Hemophilus influenza type b.
 - c. Staphylococci.
 - d. Listeria.
- 2. Viral infections (80%):
 - a. Enterovirus infection esp. echovirus type 9 (most common).
 - b. Hemorrhagic fevers: $\underline{\mathbf{B}}$ lack measles, $\underline{\mathbf{C}}$ ytomegalovirus and $\underline{\mathbf{D}}$ engue fever

4. D.D of parotid swelling

- A. Mumps:
 - <u>Pain</u> at (one or both sides) of parotid around the ear aggravated by chewing of mandible.
 - Swelling:
 - elevated ear lobule
 - behind angle of mandible
 - painful and tender
 - peak at 3 days, disappears over 3-7
 - hyperemia of stenosed duct
- B. Cervical lymphadenitis:
 - not elevated lobule
 - better felt than seen
 - firm, multiple
- C. Other parotitis causes:
 - suppurative parotitis: unilateral, high grade fever, severe pain.
 - recurrent parotitis
 - calculus duct obstruction: intermittent swelling
 - Micklucz' syndrome: dry mouth, bilateral parotid, lacrimal swelling

5. Maculopapular rash.

- 1- Common exanthems:
 - measles
 - German measles
 - roseola infantum
 - scarlet fever
- 2- Other infections: rash is sometimes present:
 - Typhoid fever
 - Infectious mononucleosis
 - Enteroviral infections
 - Parvovirus B19 (slapped cheeks)
 - Lyme disease (caused by borrelia, transmitted by ticks)
 - Erythema migrans

3- Rheumatic diseases:

- Juvenile idiopathic arthritis
- SLF
- Dermatomyositis
- Kawasaki disease

4- Skin & allergic diseases:

- Sweat rash: fine papules on the neck and trunk.
- Urticarial rash: wheals with itching
- Drug rash

6. Vesiculopustular rash.

1. Infections:

- a. Chicken pox & shingles.
- b. Herpes simplex.
- c. Coxsachie (hand and foot syndrome).
- d. Scarlet fever (bacterial).
- e. Impetigo.
- 2. Skin and allergic diseases:
 - a. Erythema multiforme (Steven Johnson syndrome).
 - b. Papular urticaria.

7. DD of diphtheria.

1. Faucial diphtheria:

Causes of tonsillar membrane: Follicular tonsillitis – infectious mononucleosis – Agranulocytosis – Leukemia

2. Laryngeal diphtheria:

Other causes of stridor

Emergency

1. Causes of stridor.

- i. Infections (croup):
- 1. Acute laryngitis.
- 2. Acute laryngo-tracheo-bronchitis.
- 3. Bacterial tracheitis.

High fever with toxemia.

4. Acute epiglottitis

Acute life-threatening illness.

- 5. Spasmodic laryngitis.
- ii. Other causes:
 - 1. Laryngospasm:

As in hypocalcemic tetany.

2. Laryngomalacia:

Recurrent or continuous stridor since birth.

- 3. Laryngeal edema:
 - With severe allergy.
 - Following extubation.
- 4. Laryngeal foreign body (peanut or toy):

Sudden onset of cough.

5. Laryngeal compression:

Retropharyngeal hematoma or abscess.

6. Laryngeal diphtheria, measles or infectious mononucleosis.

2. Causes of respiratory failure.

	Type I (Lung failure or oxygenation failure)	Type II (Pump failure or hypercapnic failure)
Causes	Respiratory distress causes:	Respiratory depression:
	- Croup.	- CNS infection.
	- Epiglottitis.	- Intracranial hemorrhage.
		- CNS depressants as
	- <u>A</u> spiration.	morphine.
	- <u>A</u> cute severe asthma.	
		Respiratory muscle
	- <u>B</u> ronchiolitis.	<u>paralysis:</u>
	- <u>B</u> ronchiolitis obliterans.	- Guillain-Barre syndrome.
	- <u>B</u> roncho-pulmonary	- Poliomyelitis.
	dysplasia.	- Myasthenia.
		- Werdnig-Hoffmann disease.
	- <u>P</u> leural effusion.	
	- <u>P</u> neumothorax.	Respiratory muscle fatigue
	- <u>P</u> neumonia.	(severe type I failure):
	- <u>P</u> ulmonary edema.	- Severe pneumonia.
	- Adult respiratory distress	- Severe RDS.
	syndrome (ARDS).	
	- Neonatal respiratory distress	
	syndrome (NRDS).	
	- Lung collapse.	
Defect	Hypoxemia.	Hypoventilation (hypercarbia).
Clinical picture	Grades of respiratory distress	Shallow irregular breathing or
Синши рини с	(see below).	apnea.
\overline{ABG}	- Low PO ₂ .	- High PCO ₂ .
	- Metabolic acidosis.	- Respiratory acidosis.
Treatment	Oxygen therapy (free flow or with positive pressure ventilation according to ABG).	Mechanical ventilation (with or without oxygen).

3. Causes of respiratory distress.

- 1. Pulmonary or airway causes (see causes of type I failure).
- 2. Heart failure.
- 3. Metabolic acidosis.
- 4. Severe anemia.

4. Causes of coma.

	Primary or structural	Secondary or metabolic
Lesion	Focal or lateralizing (except infection)	Diffuse
Causes	1. Head trauma.	1. Hypoxic encephalopathy:
	2. CNS infection.	- Respiratory failure.
	3. Vascular (infarction,	- Heart failure.
	hemorrhage).	- Shock.
	4. Tumor.	
	5. Post-epileptic.	2. Endogenous
		encephalopathy:
		- Renal failure.
		- Liver cell failure.
		- DKA.
		- Acute hypertension.
		3. Exogenous
		encephalopathy:
		Exogenous poisons
		(organophosphorus
		compounds or drugs as paracetamol).
Diagnosis	CT scan & MRI.	Lab. investigations.
Response	Not dramatic, if any.	Responsive to treatment, if diagnosed early.

5. Causes (types) of shock.

SHOCK

- 1. $\underline{\mathbf{H}}$ ypovolemic shock (most common):
 - a. Severe dehydration due to:
 - Gastro-enteritis.
 - Vomiting.
 - Diabetic keto-acidosis.
 - Diminished intake.
 - b. Acute hemorrhage (internal or external).
 - c. Severe burn.
- 2. Distributive (<u>K</u>inetic) shock:

Loss of vascular resistance and excess vasodilation.

- a. Sepsis.
- b. Anaphylaxis (drugs).
- c. Neurogenic (spinal cord trauma).
- 3. <u>Cardiogenic shock:</u>
 - a. Severe acute heart failure.
 - b. Sepsis.

- c. Any advanced shock.
- 4. **O**bstructive shock:

Mechanical obstruction of cardiac blood flow.

- a. Tension pneumothorax.
- b. Cardiac tamponade.
- 5. Septic shock:
 - Mixed form of shock, but it is mainly a distributive shock.
 - Results from activation of systemic inflammatory response (from bacterial or viral infection).

Hemodynamic parameters in different types of shock:

Type of shock	SVR	CVP	Cardiac output
Hypovolemic	↑	\	\
Cardiogenic	↑	↑	\
Distributive	<u> </u>	<u> </u>	<u> </u>

- SVR = systemic vascular resistance.
- CVP = central venous pressure.

6. Metabolic acidosis.

- A. Loss of alkali:
 - 1. Renal tubular acidosis
 - 2. Diarrhea

B. Overproduction of acids:

- 1. Arrest
- 2. Shock (severe hemorrhage severe dehydration)
- 3. Severe pneumonia: pneumonia affect O2 exchange more than CO2 exchange
 - In all of the above: hypoxia leads to anaerobic glycolysis with lactic acidosis
- 4. D.K.A (keto-acids)
- 5. Salicylates (exogenous acids aspirin): early hyperventilation with respiratory alkalosis, later on metabolic acidosis

C. Renal failure:

Reduced production of HCO₃ and reduced elimination of acids in urine

Neonatology

1. Preterm babies.

Etiology:

- 1. Maternal chronic diseases
- 2. Maternal chronic infections
- 3. Uterine anomaly
- 4. Cervical separation
- 5. Placental separation
- 6. Premature rupture of membranes
- 7. Multiple pregnancy
- 8. Fetal hydrops
- 9. Fetal distress
- 10. Fetal anomalies

2. Large for gestational age babies (LGA).

Causes:

- 1. Constitutional LGA babies
- 2. Infants of diabetic mothers
- 3. Hydrops fetalis

3. Small for gestational age babies (SGA).

Etiology:

- 1. Chronic maternal diseases
- 2. Decreased placental flow and oxygenation: hypertension, preeclampsia collagen vascular diseases, multiple gestations
- 3. Placental factors: vascular malformations, infractions, and abruption
- 4. Fetal factors: chromosomal abnormalities & congenital infections.

4. Neonatal respiratory distress.

- A. Pulmonary
 - Neonatal respiratory distress syndrome
 - Meconium aspiration
 - Transient tachypnea of the newborn
 - Pneumonia
 - Aspiration pneumonia (meconium, secretions, or milk)
 - Congenital lobar emphysema
 - Massive pulmonary hemorrhage
 - Broncho-pulmonary dysplasia
 - Diaphragmatic hernia
 - Pneumo-mediastinum

• Pneumo-pericardium

B. Extra pulmonary

a. Airway and chest wall

- Choanal atresia (bilateral)
- Pierre-Robin syndrome
- Laryngomalacia
- Trachea-esophageal fistula
- Chest wall disorders: thoracic dystrophy
- Myasthenia gravis

b. Cardiac:

- Heart failure
- Persistent fetal circulation

c. Central (Cerebral irritation):

- Cerebral hypoxia or asphyxia
- Intracranial hemorrhage
- Meningitis

d. Metabolic

- Acidosis
- Hypothermia
- Hypoglycemia
- Hyperthermia

5. Neonatal apnea.

Causes:

1. Prematurity

2. 2ry to:

- Metabolic hypoglycemia, hypocalcemia
- Temperature instability: hypothermia or hyperthermia
- Maternal drug intake e.g. magnesium sulfate or intra-partum sedatives
- Neonatal sepsis
- Intracranial hemorrhage
- Brain hypoxia e.g. RDS, (HIE)
- Neonatal seizures
- Gastro-esophageal reflux, aspiration
- With interventions e.g. suction or ETT insertion

6. Discuss Etiology, patterns & DD of neonatal seizures.

→ *Etiology:*

See before.

→ Patterns:

A. Tonic:

- Mimic decelerate/decorticate posturing.
- Sustained posture of limbs/trunk.

- Only 30% show EEG abnormalities.
- B. Myoclonic:
 - Rapid isolated muscle jerking.
- C. Clonic:
 - One limb or side jerking rhythmic at 1-4 times/second rate.
- D. Subtle:
 - Eye: staring, deviation, blinking.
 - Buccal/lingual: chew, suck, lip smacking.
 - Limbs: cycling, row, swim.
 - Systemic: apnea, BP alterations.
- **→** DD:
- A. Jitteriness:
 - No associated eye motion.
 - Stimulated by sudden movement or noise.
 - Symmetrical rapid movement of hand.
- B. Bilateral neonatal sleep myoclonus:
 - Bilateral/unilateral jerking.
 - No stimulus.
 - Occurs during sleep.
 - Involve trunk.

7. Hypothermia (neonatal cold injury).

Causes:

- 1. Cold environment, sometimes due to negligence or abuse
- 2. Inadequate drying after birth
- 3. Inadequate clothing
- 4. Sepsis
- 5. Prematurity due to:
 - Immaturity of the heat regulating center
 - Small muscle bulk
 - Large surface area of skin
 - Diminished fat insulation
 - Diminished intake
 - Associated illness

8. Neonatal bleeding

Causes:

- 1. Deficient clotting factors:
 - a. Transient deficiencies of vitamin K-dependent factors (hemorrhagic disease of the newborn)
 - b. Acquired with: DIC and liver cell failure
 - c. Inherited abnormalities of clotting factors: hemophilia A and B

- 2. Low platelet count (thrombocytopenia):
 - a. Increased destruction in the peripheral circulation:
 - ➤ Neonatal immune thrombocytopenia:
 - Isoimmune thrombocytopenia
 - Maternal immune thrombocytopenic purpura
 - Maternal lupus, drugs
 - ➤ Non-immune thrombocytopenia
 - Disseminated intravascular coagulopathy (DIC)
 - Necrotizing enterocolitis

b. Decreased production in bone marrow: in Fanconi anemia

- 3. Other causes of bleeding:
 - a. Vascular: Hypoxia, acidosis and prematurity (CNS or pulmonary hemorrhage)
 - b. Obstetric trauma as rupture of liver or spleen and cephalhematoma

Cardiology

1. Differential diagnosis of rheumatic fever.

1. Arthritis:

From other causes of arthritis (see rheumatology).

2. Carditis:

From other causes of carditis (e.g. viral myocarditis).

3. Chorea:

From other causes of chorea (e.g. drug-induced chorea).

4. Other causes of heart failure as congenital heart diseases & myocarditis.

2. Pediatric hypertension.

Causes:

- 1. Renal: chronic pyelonephritis, glomerular disease, reno-vascular disease
- 2. Aortic coarctation
- 3. Essential hypertension
- 4. Iatrogenic (steroid-induced)
- 5. Endocrine causes (Cushing, pheochromocytoma)
- 6. Increased ICP
- 7. Pain

3. Acyanotic vs cyanotic heart diseases.

Non cyanotic (80%)	Cyanotic (20%)
Maybe asymptomatic.	Onset of cyanosis is variable.
Clinical differentiation is possible.	Clinical differentiation is impossible.
Investigations are needed.	Investigations are essential.
Some may not need surgery.	Surgery is inevitable.
With left to right shunt:	With decreased pulmonary flow:
VSD (30%). PDA (5-10%). ASD (5-10%). A-V canal (2%).	Fallot tetralogy (5%). Fallot like conditions. Pulmonary atresia.
With obstructive lesion:	With increased pulmonary flow:
Pulmonary stenosis (7%). Coarctation of aorta (5%). Aortic stenosis (5%).	TGA (5%). Truncus arteriosus. Single ventricle. Hypoplastic left ventricle.

Hematology

1. Causes (classification) of anemia:

1. Decreased RBCs production

a. Dyshematopoietic anemia

- Iron deficiency anemia
- Folic acid & vitamin B12 (megaloblastic anemia)
- Vitamin C and protein deficiency
- Cu & vitamin B6 deficiency

b. Bone marrow failure

o Pure red cell aplasia

- Inherited: Shwachman- Diamond syndrome
- AR-Associated exocrine pancreatic failure
- Acquired with (Parvo v. B19)

o Aplastic anemia (→ pancytopenia)

- Congenital → e.g. Fanconi anemia
- Acquired → idiopathic or secondary to infections (hepatitis) Toxins (insecticide) irradiation

o **Infiltration of bone marrow:**

Malignant cell e.g. leukemia or metabolic cells as Gaucher cells

2. Increased RBCs destruction:

a. Hemolysis

Corpuscular defects [hereditary] (C)	Extra corpuscular (extrinsic) [acquired] (A)	
Membrane defect:	Immunologic disorders:	Non immunologic disorders:
Spherocytosis	Coombs' +ve)	
Elliptocytosis	Rh & ABO incompatibility	Sepsis
Enzyme defect:	Autoimmune hemolytic	Malaria
G6PD deficiency (A)	anemia	Wilson disease
Pyruvate kinase def.	Idiopathic	Artificial valve
<u>Hemoglobin defect:</u>	Infections (e.g. EBV, CMV &	DIC
Quantitative: Thalassemia	Mycoplasma pneumonia)	Hemolytic uremic syndrome
Qualitative: sickle cell anemia	Drug-induced (e.g. methyldopa, penicillin)	
	Collagen vascular diseases (SLE)	

b. Hypersplenism: leading to pancytopenia

- 3. Blood loss (hemorrhagic anemia)
 - a. Acute: trauma, accidents, surgery -Varices- operative (circumcision in hemophilics)
 - b. Chronic: feto-maternal transfusion- Ankylostoma- Bilhariziasis- Meckel's diverticulum-cow milk allergy

2. Microcytic hypochromic anemia:

Disease	Clinical picture	
Iron deficiency anemia	Pica.	
Beta-thalassemia trait	No response to iron.	
Chronic infection	Picture of infection.	
Sideroblastic anemia	Improve with vitamin B6.	
Lead poisoning	Manifestations of lead toxicity.	

3. Acute hemolysis:

Disease	Specific clinical picture	Specific investigation
G6PD deficiency:	History of 1st intake of beans.	Heinz bodies.
		G6PD assay.
Autoimmune hemolytic	- Drug intake.	+ve Coomb's test.
anemia (AIHA):	- Infection 2 weeks ago.	
	- Associated arthritis or skin	
	rash.	
Hemolytic uremic syndrome	- History of severe	- Thrombocytopenia.
(HIS):	gastroenteritis.	- Elevated renal function test
	- Acute renal failure.	levels.
Infection (malaria):	- Travelling to endemic area.	Blood film is diagnostic.
	- Pattern of fever.	
Sepsis:	- Toxic patient (septicemia).	- CBC: leukocytosis, shift to
	- Purpuric eruption.	left.
		- High ESR & CRP.

4. Causes of purpura:

i. Thrombocytopenic purpura: (low platelets)

1. Increased platelet destruction (Normal megakaryocytes)

a. <u>Immune:</u>

- Idiopathic throbocyopenic purpura (ITP)
- Neonatal:
- Isoimmune thrombocytopenic
- Maternal ITP
- Systemic lupus erythematosus

b. Non immune:

- DIC
- Hemolytic uremic syndrome
- Hypersplenism
- Drug induced

2. <u>Decreased platelet production (low magakaryocytes)</u>

a. Congenital:

- Thrombocytopenic with absent radius (TAR syndrome)
- Constitutional pancytopenia (Fanconi anemia)

• Thrombopoietin deficiency

b. Acquired:

- Megakaryocytic aplasia (idiopathic or 2ry to drugs)
- Aplastic anemia (idiopathic- drugs –toxin irradiation)
- Marrow infiltration (leukemia lymphoma metabolic disorders)
- ii. Non-thrombocytopenic purpura: (Normal platelets)

1. Platelet dysfunction:

- Drugs as aspirin
- Uremia
- Inherited abnormal platelets e.g. giant platelet syndrome

2. Vascular purpura:

- Infections as meningococeemia
- Vitamin C deficiency (Scurvy)
- Inherited: Ehlar Danlos syndrome Marfan syndrome
- Immune vasculitis (HSP)

5. Differential diagnosis of ITP:

1) Aplastic anemia:

Pancytopenia & decreased all precursors in bone marrow.

2) Acute leukemia:

Hepatosplenomegaly & infiltration of bone marrow by blast cells.

3) Other causes of thrombocytopenia (discuss).

For more details:

	ITP	Aplastic anemia	Leukemia
History:	Fever 2 weeks before onset of purpura.	 History of exposure to bone marrow depressant drugs or viral infection. The cause maybe idiopathic. Fever (infections). Repeated blood transfusion. 	- Prolonged fever.- Arthralgia or arthritis.- Recent significant weight loss.
Examination:	Good general condition.No anemia except in severe blood loss.No Organomegaly.	- Bad general condition.- Marked pallor.- No Organomegaly.	- Bad general condition.- Pallor.-Hepatosplenomegaly & lymphadenopathy.
Investigations:	Blood picture: Thrombocytopenia.	Pancytopenia.	Thrombocytopenia, anemia. WBCs: normal, increased or decreased.

Anti-platelet antibodies (60%).		
Bone marro examination or increased megakaryoc defective bu	n: normal aplasia. ytes with	Infiltration by blast cells.

6. DD of hemophilia in general:

- 1. Acquired coagulation defects as liver failure: clinical & laboratory evidence of LCF.
- 2. Disseminated intravascular coagulation (DIC): critically-ill patient, fibrin degradation products (FDPs) in blood.

7. Differential diagnosis for children with anemia and splenomegaly.

- 1. Chronic hemolytic anemia: (but SCA patients will develop auto splenectomy + crisis)
 - Thalassemia
 - spherocytosis
- 2. Leukemia.
- 3. Gaucher disease.
- 4. Iron deficiency anemia (splenomegaly in 15% of cases).
 - Bilharziasis (portal HTN & hypersplenism + bleeding)
- 5. RH incompatibility (hemolytic disease of the newborn)
- 6. Wilson disease (cirrhosis \rightarrow portal HTN \rightarrow splenomegaly)
- 7. Malaria.
- 8. Sepsis.
- 9. Causes of portal HTN leading to splenomegaly → hypersplenism
- 10. Chronic autoimmune anemia (cirrhosis \rightarrow portal HTN \rightarrow splenomegaly)
- 11. Patients with cirrhosis:
 - Bleeding tendency → anemia
 - Portal HTN → splenomegaly

8. Generalized purpuric eruptions + recent blood transfusion + pallor:

- A. With splenomegaly:
 - Complicated Chronic hemolytic anemia (with megaloblastic crisis _ aplastic crisis hyperhaemolytic crisis [associated G6PD def] hypersplenism)
 - o Leukemia or lymphoma
 - Storage disease
- B. No splenomegaly:
 - o Active bleeding in: ITP Henoch Schonlein purpura leukemia TAR \$
 - o Eban\$
 - o Aplastic anemia (Fanconi anemia)

Respiration

1. DD of wheezing in infancy and childhood.

DD of asthma.

- A. Acute non recurrent wheezing (wheezing for the 1st time):
 - 1. Acute bronchiolitis (**commonest** cause of wheezing in **infants**).
 - 2. Severe bronchopneumonia with generalized obstructive emphysema.
- B. Chronic persistent or recurrent wheezing:
 - 1. Bronchial asthma (**commonest** cause of wheezing in **children**):

Never diagnose asthma from the 1st attack.

- 2. Chronic or recurrent infections:
 - Cystic fibrosis.
 - Immunodeficiency.
- 3. Recurrent aspiration as in GERD.
- 4. Foreign body inhalation:
 - Sudden onset of wheezing.
 - No response to bronchodilators.
- 5. Congenital anomalies.
- 6. **C**ompression of airways:
 - Cysts.
 - Enlarged LNs.
 - Tumors.

2. DD of cough.

→ *Definition:*

- The most common symptom of respiratory disease.
- Caused by irritation of nerve receptors in pharynx, larynx, trachea and bronchi.

→ *Etiology:*

1. Acute cough (duration less than 2 weeks):

Without respiratory distress:

- a. Acute bronchitis.
- b. Acute laryngitis.
- c. Acute sinusitis.

With respiratory distress:

- a. Acute bronchiolitis.
- b. Pneumonia.
- c. Acute asthmatic attack.
- 2. Prolonged cough (duration between 2 weeks and 2 months):
 - a. Complicated bronchitis:
 - Bacterial bronchitis.
 - Segmental collapse.
 - Pneumonia.

- b. Sinusitis (due to postnasal discharge).
- c. Pertussis (whooping cough) & pertussis-like illness.
- 3. Chronic cough (duration more than 2 months):
 - a. Chronic infections:
 - Pulmonary tuberculosis.
 - Bronchiectasis.
 - b. Chronic or persistent asthma.
 - c. Recurrent aspiration.

3. DD of pertussis (whooping cough).

Adenovirus infection (pertussis-like illness):

- Less marked lymphocytosis.
- Vaccinated child.

4. DD of recurrent chest infection

- **→** Symptoms:
 - Chronic wheezy chest
 - Chronic cough (except muscular)
- → Causes:
 - 1. Cystic fibrosis
 - 2. Immotile cilia syndrome
 - 3. Cardiac cases
 - 4. Immunodeficiency
 - 5. Recurrent aspiration:
 - GERD
 - TEF
 - CP
- 9. Muscular:
 - Down
 - Duchenne
 - Rickets

5. Indications of corticosteroids in TB.

- 1. Allergy
- 2. Ascites
- 3. Bronchial
- 4. **B**rain
- 5. Cervical LN
- 6. **D**issemination

6. Pneumonia organisms based on age

- A. Neonates: (same causes as neonatal sepsis)
 - 1. GBS (commonest)
 - 2. Chlamydia
 - 3. Listeria
 - 4. Enteric bacteria (intrauterine and postnatal)
- B. Infant:
 - 1. RSV (commonest of all)
 - 2. Pneumococci (commonest bacteria)
- C. School age:
 - 1. Mycoplasma (commonest) \rightarrow mainly present with hemolytic anemia.

7. Pneumonia organisms based on type of patient.

- A. Hospitalized:
 - 1. Pseudomonas
 - 2. Candida
 - 3. Staph
 - 4. CONS
- B. Cystic fibrosis:
 - 3. Aspergillus
 - 4. Pseudomonas
 - 5. Staph
 - 6. Hemophilus
- B. Immunocompromised:
 - 1. TB
 - 2. Aspergillus

GIT

1. DD of acute abdominal pain:

A. Acute abdominal infection:

(Diagnosis: fever-systemic manifestation + the site of pain and tenderness)

- 1. Streptococcal pharyngitis
- 2. Acute gastroenteritis
- 3. Acute hepatitis
- 4. Acute appendicitis
- 5. Acute pyelonephritis
- 6. Acute pancreatitis
- 7. Acute peritonitis
- B. Acute medical conditions:
 - 1. Henoch-Schonlein vasculitis
 - 2. Sickle cell anemia
 - 3. Right lower lobe pneumonia
 - 4. Acute rheumatic fever
 - 5. Diabetic ketoacidosis
 - 6. Drug intoxication
 - 7. Lead poisoning
- C. Surgical causes:

(Multiple Air-fluid level/Bilious vomiting/complete Constipation/severe Distention)

- 1. Acute intestinal obstruction:
 - a. Incarcerated inguinal hernia
 - b. Intussusception (imp.)
 - c. Volvulus
 - d. Impacted fecal masses
 - e. Round worm masses
- 2. Other surgical causes of acute abdomen:
 - a. Inflamed Meckel's Diverticulum
 - b. Acute appendicitis
 - c. Renal stone
 - d. Gall bladder stone

2. Recurrent abdominal pain:

	Dysfunctional recurrent abdominal pain "nonspecific" or "psychogenic"	Organic recurrent abdominal pain	
Definition	Pain that does not interfere with the child activity or general health	Pain that interrupt child normal activity and health	
Incidence	>90%	Less than 10% of cases	
Pain	Periumbilical, Non localized Vague, not severe Subsides spontaneously in less than 20 minutes	Localized away from umbilicus Severe Does not improves spontaneously	
Association	No	Diarrhea- constipation- rectal- bleeding- hematuria- dysuria	
Signs	Child appear healthy No mass or tenderness	Weight loss, anemia, weight loss Organomegaly or locl tenderness	
Causes	Stressful events: loss of a parent, delivery of new sibling, school phobia Sympathy gaining Stimulate an adult with recurrent abdominal pain	Common causes: Parasitic infections: Giardiasis, amoebiasis Chronic constipation Bad selection of food Lactose intolerance (disaccharide) Chronic use of drugs Renal calculi Familial Mediterranean fever	
Simple investigations	Normal	Abnormal	
	Urine –stool- CBC – abdominal x ray		
Further inv.	Not needed	According to the clinical suspicious	
Treatment	Reassurance	according	

3. DD of vomiting in infancy:

- A. Dietetic errors:
 - 1. Over feeding
 - 2. Excess carbohydrate in diet
 - 3. Irregular feeding or tight abdominal binder
- B. Infection:
 - 1. Gastro-enteritis

- 2. Appendicitis
- 3. Urinary tract infection
- 4. Respiratory infection: otitis media, whooping cough or pneumonia
- 5. CNS infections: Meningitis, encephalitis or brain abscess

C. Medical disorders:

- 1. Celiac disease
- 2. Renal failure
- 3. Metabolic disease
- 4. Raised cranial tension
- 5. Diabetic ketoacidosis

D. Intestinal obstruction:

- 1. Intussusception
- 2. Volvulus
- 3. Adhesions
- 4. Strangulated inguinal hernia
- 5. Foreign body

4. Chronic abdominal masses:

- 1. Hepatosplenomegaly (most common)
- 2. Renal and suprarenal masses:

Include: Wilms tumor, neuroblastoma, hydronephrosis, renal vein thrombosis, polycystic kidney disease and cystic dysplastic kidney

	Neuroblastoma	Wilms tumor		
Origin	From the suprarenal	From the kidney		
Onset	Below the age of 3 years	Around the age of 3 years		
Mass site	Right or left upper quadrant mas	Right or left upper quadrant mass		
Mass character	Hard with irregular surface	Firm with a smooth surface		
	Cross the middle line	Doesn't cross the midline		
Other manifestation	Hepatomegaly- proptosis- anemia- subcutaneous nodules Hematuria may be also present			
Investigations	Bone marrow biopsy: neuroblastoma cells in 70% of cases.	CT abdomen and biopsy		

- 3. Pancreatic masses: (non mobile)
 - Pancreatic pseudo cyst: (the most common pancreatic mass due to blunt trauma)
 - Pancreatic cystadenoma
 - Retention cyst
- 4. Intestinal masses: (mobile midabdominal)
 - Intestinal cysts: Mesenteric cyst & omental cyst
 - Intestinal lymphoma: may be very huge- associated with ascites
 - Intestinal inflammatory masses: Tuberculous mesenteric adenitis

- 5. Retroperitoneal masses: (upper or lower abdomen)
 - Include teratoma, Rhabdomyosarcoma and lymphoma
- 6. Masses in females:
 - Ovarian cysts and tumors
 - Uterine causes
 - Tumors or hemato-metra
 - Vaginal: hematocolpos- tumors

5. DD of Diarrhea:

I. Acute diarrhea:

- A. Infective diarrhea (gastroenteritis): most common cause of diarrhea in infant and children
 - 1. Viral agents: Rotavirus-enterovirus-adenovirus.......
 - 2. Bacterial agents: campylobacter jujeni- shigella salmonella- E. coli......
 - 3. Parasitic agents: Entamoeba histolitica and giardia lamblia
- B. Non-infective diarrhea:
 - 1. Dietetic diarrhea: overfeeding or inappropriate food for age
 - 2. Drug- induced diarrhea: Antibiotics especially oral ampicillin
 - 3. Parenteral diarrhea

II. Chronic diarrhea:

- A. Chronic GIT infection:
 - 1. TB
 - 2. Giardiasis
- B. Malabsorption syndromes
 - 1. Cholestasis
 - 2. Cystic fibrosis
 - 3. Achlorhydria
 - 4. Celiac disease
 - 5. Short bowel syndrome
 - 6. Lymphangectasia
 - 7. Inflammatory bowel disease

III. Persistent diarrhea:

Causes:

- 1. Sugar intolerance.
- 2. Cow's milk protein allergy.
- 3. Overgrowth of bacteria in the upper small intestine.
- 4. Mucosal injury and atrophy.

6. DD of dehydration:

- 1. Gastroenteritis: isotonic dehydration.
- 2. DKA, high fever, hot environment, excessive sweating: hypertonic dehydration.
- 3. Prolonged diarrhea with compensation by drinking water or hypotonic solution: hypotonic dehydration.

Dehydration	Isotonic	Hypertonic	Hypotonic
Skin:	Poor turgor	Fair turgor	Very poor turgor
Eye:	Sunken	Mildly sunken	Very sunken
CNS:	Normal	Irritability & seizures	Lethargy & coma
Tongue:	Normal	Dry	Moist
Serum Na ⁺ :	Normal	> 150	< 130

7. Causes of hematemesis.

- 1. Swallowed blood
- 2. Esophagitis
- 3. Esophageal varices
- 4. Gastritis
- 5. Peptic ulcer
- 6. General causes of bleeding (ITP, hemophilia)

8. Causes of bleeding per rectum.

$\underline{All\ of\ hematemesis}\ +$

- 1. Necrotizing enterocolitis
- 2. Hemorrhagic disease of the newborn
- 3. Intussusception
- 4. Henoch shenolein purpura
- 5. Infection: amoebiasis bacillary desentry
- 6. Inflammatory bowel disease
- 7. Anal fissure
- 8. Piles

Hepatology

1. Causes of hepatomegaly.

- 1. Storage:
 - a. Fat: malnutrition, obesity, cystic fibrosis, metabolic liver disease.
 - b. Lipid storage disease: Niemann pick or Gaucher disease.
 - c. Glycogen: glycogen storage disease or infant of diabetic mother.
 - d. Others: as alpha-1 antitrypsin deficiency, Wilson's disease, Schistosomiasis.
- 2. Inflammation:
 - a. Acute or chronic viral hepatitis.
 - b. Autoimmune hepatitis.
 - c. Liver abscess.
- 3. Infiltration:
 - a. Cystic: choledochal cyst.
 - b. Malignant: hepatoblastoma or hepatocellular carcinoma.
 - c. Metastases: neuroblastoma, histiocytosis, lymphoma, leukemia.
- 4. Increased size of vascular spaces:
 - a. Budd-Chiari syndrome.
 - b. Hepatic veno-occlusive disease (VOD).
 - c. Right sided heart failure.
 - d. Constrictive pericarditis.
 - e. Restrictive cardiomyopathy.
- 5. Increased size of biliary spaces:
 - a. Biliary obstruction: atresia.
 - b. Congenital hepatic fibrosis.

2. Causes of hepatosplenomegaly.

1) Neonatal period and early infancy:

Causes of cholestasis (enumerate).

- 2) Late infancy and early childhood: (1-6 years)
 - a. Chronic hemolytic anemia (Mediterranean): spleen is enlarged more.
 - b. $\underline{\mathbf{M}}$ etabolic diseases: Gaucher, Niemann-Pick, glycogen storage disease, mucopolysaccharidosis, ..
 - c. <u>M</u>alignancy: acute leukemia (anemia, purpura & hepatosplenomegaly).
- 3) Late childhood: (Above 6 years)
 - a. Chronic hepatitis and post-hepatitic cirrhosis.
 - b. Metabolic: Wilson's disease.
 - c. Bilharziasis.
 - d. Autoimmune.

3. Causes of hepatomegaly without portal hypertension.

- 1. Chronic myeloid leukemia
- 2. Polycythemia
- 3. Chronic malaria
- 4. Typhoid fever
- 5. Brucellosis
- 6. Epstein par virus
- 7. Amyloidosis, hemochromatosis
- 8. Leukemias
- 9. Lymphomas
- 10. Glycogen storage and lipid storage diseases

Nephrology

1. DD of hematuria:

A. Non-glomerular:

- 1. Infection (bacterial-viral TB-schistosomiasis) (the commonest cause in children)
- 2. Trauma to genitalia, urinary tract or kidney
- 3. Tumors
- 4. Stones or hypercalciuria
- 5. Cytotoxic drugs
- 6. Exercise
- 7. Sickle cell disease or any cause of renal infarction
- 8. Renal vein thrombosis

B. Glomerular:

- 1. Acute glomerulonephritis
- 2. 2^{ry} nephritis as in: Henoch Schonlein purpura-polyarteritis nodosa- systemic lupus erythematosus-wegners granulomatosis
- 3. Familial nephritis as in Alport syndrome

C. Bleeding tendency:

- 1. ITP
- 2. Hemophilia
- 3. Hepatic failure

2. DD of polyuria:

Causes:

- 1. Diabetes mellitus
- 2. Hypervitaminosis D
- 3. Chronic renal failure
- 4. Diabetes insipidus
- 5. Renal tubular disorder
- 6. Psychogenic polydipsia

3. DD of oliguria:

Pathophysiology	Clinical consequence	
Fluid retention	Edema & weight gain	
Hypertension	Congestive heart failure-pulmonary edema	
Retention of acidic wastes	Metabolic acidosis: Acidotic breathing (rapid and deep)	
Water retention	Dilutional hyponatremia- dilutional hypocalcaemia	
Retention of K ⁺	Hyperkalemia-arrhythmia	
Retention of urea and creatinine (Azotemia)	Hyperosmolarity-uremic encephalopathy &	
	coma-brain edema	

4. Enuresis.

- A. Primary causes:
- 1. Delay in maturation of bladder control has been postponed.
- 2. Genetic component: a family history is found in most children.
- 3. Children show deeper sleep and difficulties in waking
- 4. Loss of the normal nocturnal rise in anti-diuretics hormone (ADH) production.
- B. Secondary causes:
- 1. DM
- 2. Diabetes insipidus
- 3. UTI
- 4. Stones

Neurology

1. DD of acute paralysis in children:

- **→** *Etiology:*
- 1. Spinal cord:
 - Transverse myelitis.
 - Spinal cord trauma (as in road traffic accidents).
- 2. Anterior horn cells:
 - Poliomyelitis: asymmetric ascending paralysis.
- 3. Peripheral nerves:
 - Guillain-Barre syndrome (commonest cause): symmetric ascending paralysis.
 - Post-diphtheritic paralysis: **symmetric descending** paralysis.
- 4. Neuromuscular:
 - Botulism: **symmetric descending** paralysis.

2. Causes of mental retardation.

1. Hereditary causes:

a. Chromosomal disorders:

- Triosmy & monosomy e.g. Down syndrome Edward syndrome
- Sex chromosomal abnormality e.g. fragile x syndrome
- Structural chromosomal abnormalities (deletion as in Brader Willi syndrome)

b. Genetic autosomal dominant micorcephaly

Autosomal dominant microcephaly

c. Inborn errors of metabolism:

- Amino acids e.g. phenyl ketonuria, tyrosinemia
- Carbohydrate e.g. galactosemia

d. Neurodegenerative disorders:

Lysosomal disorder: Lipidosis & mucopolysaccharidosis

e. Neurocutaneous disorders:

Neurofibromatosis- Tuberous sclerosis- Sturge Weber syndrome

2. Acquired causes:

a. Prenatal causes

- Congenital malformations: hydrancephaly, porencephaly
- Congenital infections: CMV, rubella, toxoplasmosis [TORCH]

b. Natal causes

- Hypoxic ischemic syndrome
- Birth injuries as intracranial hemorrhage
- Prematurity

c. Postnatal causes

- Trauma: accidental or non-accidental
- Infections: Encephaliti, meningitis
- Hypoxia: asphyxia, status epilepticus

- Metabolic and: Hypoglycemia & hypernatremia
- Endocrine: hypothyroidism
- Poisoning: lead poisoning

3. Seizures.

- A. Epilepsy: (Recurrent seizures unrelated to fever or acute cerebral insult)
 - 1. <u>Idiopathic (80%)</u>
 - 2. **Secondary (20%)**
 - Cerebral deformation / malformation / vascular occlusion
 - Cerebral damage:
 - Congenital infections
 - Hypoxic- ischemic encephalopathy
 - Intraventricular hemorrhage
 - Cerebral tumor
 - Neurodegenerative disorders
 - Neurocutaneous syndromes
 - Tuberous scelorosis
 - Neurofibromatosis
 - Sturge Weber syndrome
- B. Non-epileptic:
 - 1. Febrile convulsions
 - 2. Brain edema
 - 3. Metabolic: hypoglycemia hypocalcaemia- hypomagnesaemia- hyponatremia hypernatremia
 - 4. Head trauma
 - 5. Meningitis and encephalitis
 - 6. Poisons and toxins

4. Floppy infant.

Etiology:

- 1. Central (Cerebral disorders)
 - Atonic cerebral palsy
 - Cortical malformations
- 2. Genetic syndrome
 - Down syndrome
 - Prader-Willi syndrome: delayed development- delayed growth obesity hypotonia
- 3. Neuromuscular disorders:
 - Anterior horn cells: spinal muscle atrophy: Werding Hoffmann disease
 - Hereditary neuropathy
 - Neuromuscular junction: Transient neonatal myasthenia
 - Congenital myopathy

5. Progressive motor weakness.

Causes:

1. Brain causes:

- Brain tumors and cysts
- Degenerative brain diseases

2. Spinal cord causes:

- Compression paraplegia (Spinal cord tumors, potts disease)
- Degenerative diseases of the spinal cord

3. Nerve:

Hereditary motor sensory neuropathy

4. Neuromuscular junction:

Myasthenia gravis

5. <u>Muscular causes:</u>

Muscular dystrophies (Duchenne muscular dystrophy is the most common cause of progressive weakness)

Allergy

Causes of utricaria:

- 1. Idiopathic (common)
- 2. Infection
- 3. IgE-mediated
 - Specific food-cow's milk, nuts (especially peanuts), fish
 - Blood products
 - Drugs –penicillin, cephalosporin
- 4. Pharmacological
 - Foods containing histamine -releasing substances, e.g. strawberries, egg white
 - Aspirin and other non-steroidal anti-inflammatory agents
- 5. Physical agents: (heat, cold, pressure)

Rheumatology

1. Arthritis.

- A. Congenital:
 - Cystic fibrosis
- B. Inflammatory:
- 1. infective:
- a. acute:
- bacterial (pyogenic)
- viral (rubella, mumps)
- b. chronic:
- ТВ
- 2. non-infective:
- a. post. Infectious:
- rheumatic
- reactive
- b. collagen diseases:
- JIA
- SLE
- PAN
- c. vasculitis:
- HSP
- Kawasaki
- d. inflammatory bowel disease:
- crohn's
- ulcerative colitis
- C. neoplastic:
- 1- leukemia
- 2- neuroblastoma
- 3-lymphoma
- D. traumatic
- E. others: hematological:
- SCA
- hemophilia

Endocrine

1. Precocious puberty.

Definition:

The appearance of 2ry sexual characters < 8 yr in girls and < 9 yr in boys

Types and causes:

1. Normal variant:

a. Premature thelarche:

Early breast enlargement without other signs of puberty (Between 6 months & 3 years)

b. Premature adrenarche: (premature pubarche)

Definition:

Pubic hair develops before age of puberty

Pathogenesis:

Premature maturation of supra-renal androgens

Investigations:

To exclude central precocious puberty

c. Gynecomastia in males:

Breast enlargement in boys

It may be a sign of puberty, local causes or hormonal causes.

2. Precocious puberty:

A. True precocious puberty (gonadotropin dependent)

- Gonadotropin level is high (prebertal)
- Gonads are enlarged (testes in males and ovaries in females)
- Spermatogenesis occurs in males and ovulation occurs in females

• The main causes

- Idiopathic: 80% of cases in females and 50% of cases in males
- Organic causes: secondary to CNS tumors, hydrocephalus, trauma and radiotherapy. It is commoner in males.

B. Pseudoprecocious puberty (gonadotropin independent)

- Gonadotropin level is low (prepubertal)
- Gonads do not enlarge
- Spermatogenesis in males and ovulation in females do not occur

• Causes:

- In females: ovarian tumors or excess estrogen
- In males: testicular tumors or excess androgens

2. Delayed puberty.

Definition:

Delayed secondary sexual characters beyond 13 yr in girls & 14 yr in boys

Types and causes:

1. Constitutional delay of growth and puberty/ familial: (the commonest)

2. Low gonadotropin secretion (hypogonadotropic hypogonadism):

- Systemic disease: as Cystic fibrosis, Crohn's disease, organ failure, Anorexia nervosa
- Starvation, excess physical training
- Hypothalamopituitary disorder
- Pan-hypopituitarism
- Isolated gonadotropin deficiency
- Intracranial tumors (including craniopharyngioma)

3. High gonadotropin secretion (hyper-gonadotropic hypogonadism):

- Chromosomal abnormalities:
 - Klinefelter syndrome (47 XXY)
 - Turner's syndrome (45 XO)
- Steroid hormone (androgens- estradiol) enzyme deficiencies
- Acquired gonadal damage:
 - Chemotherapy
 - Radiotherapy
 - Trauma
 - Torsion of the testis

COLLECTIONS

1. Immunosuppressive:

- Aplastic Anemia: ATG, Cyclosporine, others
- Chronic ITP: Cyclosporine, Azathioprine
- Autoimmune Hepatitis: Azathioprine
- SLE: Azathioprine, Cyclophosphamide
- Steroid Resistant NS: Cyclophosphamide, Mycophenolate

2. Post-infectious Sequel (Immune mediated):

- Upper Respiratory Tract infection: Rh. F., MCNS, Post Strept GN, ITP, HSP, Asthma (Intrinsic)
- Gastroenteritis: Guillen Barre
- (Viruses related to DM I?)

3. Desmopressin:

- Hemophilia A
- vWF deficiency.
- Enuresis
- Esophageal Varices

N.B. in OBGYN? Fibroid, intraoperative to decrease bleeding.

4. Chorea:

- Rheumatic Fever
- Wilson's Disease
- CP (dyskinetic)

5. Attacked by Capsulated Organisms:

- Splenectomy
- Nephrotic Syndrome
- Bacterial Meningitis in Children above 2 months

6. Toxic Clubbing:

- Infective endocarditis
- Lung Abscess
- Bronchiactesis

N.B. another name? Pale Clubbing. Cyanotic Clubbing is also called Blue Clubbing.

7. "Fleeting"

- Arthritis in Rheumatic Fever
- Rash in systemic onset JIA

8. Non Chest symptoms in Pneumonia:

- Abdominal Pain (lower lobe)
- Neck Pain

9. Bacterial Lymphocytosis:

- TB
- Pertussis

10. "Absolute" in labs:

- Lymphocytosis: Pertussis
- Neutropenia: Neonatal Sepsis

11. Thrombosis:

- Congenital Cyanotic Heart Disease
- Nephrotic Syndrome
- SCA

12. Infections predispose to:

- 2 Crises in SCA (VO and Inf.)
- Acute Hemolysis in G6PD
- Hyper cyanotic Spell in Fallot

13. ACE-I:

- Diabetic Nephropathy
- Ped HTN (Stage <u>2</u> or <u>2rv</u>)
- CKD
- Nephrotic Syndrome

14. More common in MALES:

- MCNS (2:1)
- HSP
- CHPS
- BA (2:1)
- Febrile Convulsions
- Enuresis
- All X linked diseases as Duchenne, hemophilia a &b

15. More common in FEMALES:

- ASD
- PDA
- Chorea in Rheumatic Fever
- Acquired hypothyroidism
- SLE

16. Alopecia:

- SLE
- S.E. of Cyclophosphamide

17. Projectile Vomiting:

- Increased ICT
- CHPS

18. Hyperventilation:

- <u>Diagnostic:</u> Triggers absence seizure
- <u>Therapeutic:</u> Brain edema

19. Ankylosis

- Hemophilia
- JRA
- CP

20. Crohn's

- Arthritis
- Recurrent abdominal pain (and bleeding per rectum)
- Delayed Puberty

21. Splenectomy is curative in

- Spherocytosis
- Chronic ITP

22. Hepatitis

- Aplastic Anemia
- 2ry Nephrotic/Nephritic Syndrome
- Type 1 DM

23. EBV

- Hemolytic Anemia (IMMUNE)
- BM Depression

24. Chemical Toxins

- Type 1 DM
- Aplastic Anemia

25. Maternal SLE:

- Complete Heart Block
- Neonatal Thrombocytopenia

• Preterm and SGA

26. Mottled Skin:

- Shock
- Severe Dehydration
- Cong. Hypothyroidism

27. Mycoplasma:

- Pneumonia (School age)
- Hemolytic Anemia (IMMUNE)
- Meningitis

28. Pericardial Effusion:

- Systemic onset JRA
- SLE
- Rh Carditis

29. Activation of TB:

- Pertussis
- Measles

30. ↓ Anticoagulants:

- Ptn S def: Neonatal seizures, Chicken pox (Purpura Ful.)
- Loss of Antithrombin: Nephrotic Syndrome

31. Mucopolysaccharidosis:

- Macrocephaly (<u>Hurler's</u>)
- Disproportionate Short Stature (Morquio's)
- Mental Retardation (Neurodegenerative Disorders)
- Epilepsy (Neurodegenerative Disorders)
- X-Linked recessive (Hunter's)
- HSM

32. Parvovirus:

- Slapped cheek syndrome (DD of Maculopapular rash)
- RBC Aplasia (Anemia)
- Aplastic Crisis (Chronic Hemolytic Anemia)

N.B. Parvovirus causes NON-IMMUNE Hydrops in fetus. (OBGYN)

33. Hyaline:

- Membrane Disease (RDS)
- Testis (Klinefelter)

34. "400,000" in Infections:

- Vit A dose in Measles
- Oral Penicillin V dose in Scarlet fever

35. Drugs given Slowly IV:

- Ca Gluconate
- Diazepam
- Phenobarbitone
- Phenytoin
- Bicarbonate
- Aminophylline

36. Drugs given ET:

- Adrenaline: Emergency, Neonatal Resuscitation
- Surfactant: RDS

37. Phenobarbitone:

- Anticonvulsant (1st line in neonatal seizures)
- Criggler Najjar II

38. Aspirin:

- مشاكل: Reye Syndrome, Acidosis, BIND, G6PD, Purpura
- Ttt: Rh Fever (Arthritis and mild carditis), HSL, Fallot, Nephrotic Syndrome

39. Chloramphenicol:

- G6PD (Acute hemolysis)
- Aplastic anemia
- Contraindicated in Breastfeeding

40. Adrenaline Doses:

- 0,1 IV or 0,3 ET (Emergency)
- 0,3 or 0,5 ET (Neonatology)
- 0,01 IM (Anaphylaxis)

41. IVIG:

- ITP (1gm/kg/day) 2 days
- Rh incompatibility (1gm/kg) over 2 hours
- Guillain Barre

42. Indomethacin:

- Contraindicated in Breastfeeding
- ttt of PDA

N.B. Causes Oligohydramnios & may be used as tocolytic in PTL & in ttt of Polyhydramnios in OB/GYN.

43. Theophylline:

- <u>ttt</u>: Neonatal Apnea and Asthma
- Cause: Neonatal Seizures

44. Eye manifestations:

- Kerato- & Lenticonus (Alport Syndrome in Neph. Syndrome)
- Cataract (Rubella and Galactosemia)
- Chorioretinitis (CMV and Toxoplasma)
- HSV (Lid, Conj and Corneal affection)
- Microphthalmia (Fanconi & Patau)
- Exophthalmus (Crouzon's [craniostenosis])
- Allagile (Corneal Affection)
- Osteogenesis imperfecta (Blue Sclera)
- Oligoarticular JRA (Chronic Iridocyclitis)
- Neuroblastoma (Proptosis)
- Pertussis (Subconj. Hge)
- Dehydration (Sunken)
- Mysthenia (Ptosis)
- Hydrocephalus (Sunset)
- Squint (increased ICT, Hydrocephalus and Down Syn)

45. Ear manifestations:

- Poor recoil in Preterm
- Osteogenesis imperfecta (poor hearing)
- Alport syndrome (poor hearing)
- Down syndrome (malformation & poor hearing)
- Turner syndrome (poor hearing)

46. Viruses with high fever:

- Measles
- Diabetes
- Herpes gingivostomatitis

47. After week 34 (late preterm):

- No Apnea
- Good surfactant
- Less risk for ICHge

48. Periventricular:

- Calcifications in CMV
- Leukomalacia in Preterm

49. Renal Vein Thrombosis:

- Acute Kidney Injury
- Hematuria
- Chronic Abdominal Mass
- Complication of neonatal Polycythemia

50. Enteroviruses:

- Polio
- Coxsackie: <u>Hand & Foot syndrome</u>, <u>Encephalitis</u>, <u>Herpangina</u>, <u>Myocarditis</u>, <u>associated</u> <u>with DM I.</u>
- Echo: Encephalitis, Fever with Purpura (Echovirus 9)

51. Erythema:

- Migrans: Lyme disease
- Marginatum: Rh. F.
- Nodosum: Complications of Scarlet Fever
- Infectiousum: Parvovirus
- Multiforme: Steven Johnson Syndrome
- Toxicum: Neonate (reaction to milk)

52. Tenesmus:

- Amoebic
- Bacillary
- TB
- Ulcerative Colitis

53. CMV:

- Type I DM
- Congenital Inf
- Encephalitis
- Hemolytic Anemia
- Rash + Fever
- Prolonged Fever
- Contraindication of Breastfeeding

54. Opisthotonus:

- Kernicterus
- Tetanus
- Meningitis

• & generally, an antigravity position in UMNL

55. "Giant":

- Giant Platelet Syndrome
- Gigantism
- Soto's Syndrome (Macrocephaly)
- <u>Giant</u> cell hepatitis (Cholestasis)

56. Exchange Transfusion:

- Neonatal hyperbilirubinemia
- VOC in SCA (if stroke, chest, priapism)
- Neonatal Polycythemia (Partial Exchange Transfusion)
- Fallot (Partial Exchange Transfusion)

57. Antibiotics for 4-6w:

- Inf. Endocarditis
- Suppurative Lung Disease
- Brain abscess
- Septic Arthritis in Neonatal Sepsis

58. Large Joint Affection:

- Rh. F.
- TB
- JIA

59. Found in urine:

- Copper in Wilson's
- Succinyl Acetone in Tyrosinemia
- Reducing Substance in Galactosemia
- Phenyl ketones in Phenylketonuria

60. "Bronzed":

- Diabetes (Hemosiderosis)
- Baby Syndrome (if Cholestasis is treated with Phototherapy)

61. Steroids:

- HSP (GIT, Nephritis, ICHge)
- Aplastic Anemia (Fanconi)
- Acute Lymph. Leukemia (<u>Prednisone</u> -> Remission, <u>Hydrocortisone</u> -> Intrathecal)
- ITP (2mg/ kg Prednisone)
- Rh. Carditis (2 mg/ kg Prednisone)
- Nephrotic Syndrome (2mg/ kg Prednisone)
- SRNS (Methylprednisolone)

- SLE (Prednisone oral, Methylprednisolone IV in acute exacerbation)
- JRA (Oral if systemic onset + Pericarditis)
- Asthma (Severe attack -> IV hydrocortisone, Moderate attack -> Prednisone, Control -> Inhaled & Oral)
- Eczema
- Allergic Rhinitis
- Anaphylaxis
- TB (if ABCD: Ascites, Allergic reaction to drugs, Bronchial Spread, Cervical LN removal, Dissemination -> Meningitis and Miliary)
- Antenatal in RDS (Beta. or Dexa.)
- Autoimmune Hepatitis (Chronic)
- Hypervitaminosis D •
- Meningitis (with H. influenzae)
- Infantile Spasms (+ ACTH)
- Coma (Dexamethasone as brain dehydrating agent)
- Duchenne's (10 days per month)

62. Adrenaline doses.



أصلا مع نفسه Anaphylaxis is 0.01 IM

63. Antibiotics in Respiratory system

- معظم العلاج في الشابتر ٧-١٠ ايام

- ولو هدى antibiotic غالبا هيبقي antibiotic

- 50 mg/Kg
- 1. OM
- 2. strept tonsillitis
- 3. bronchitis
- 4. pneumonia → + gentamycin
- 5. infective endocarditis prophylaxis

64. Clinical diagnosis:

- 1. HSP
- 2. Scarlet fever
- 3. Guillain Barré syndrome
- 4. Pancreatitis of mumps
- 5. (Prolapse in gyn.)

65. Causes of Intra-cranial hge:

- 1. Coarctation of aorta
- 2. ITP, HSP & hemophilia
- 3. Birth injury
- 4. (Eclampsia in obs.)

66. Conditions following viral URT infection:

- 1. ITP
- 2. HSP
- 3. MCNS & PSGN

67. Conditions following GIT infection:

- 1. Guillain Barré syndrome (following campylobacter pylori infection)
- 2. HUS (following E. coli infection)

68. Rash starts on:

- 1. Face \rightarrow Measles & German measles
- 2. Trunk → Roseola & chickenpox
- 3. Between them = Base of the neck \rightarrow Scarlet fever

69. No:

- 1. HF in Fallot's tetralogy (the 2 ventricles support each other)
- 2. IE in ASD (low P gradient)

70. -ve hyperoxia test:

- 1. TGA
- 2. Eisenmenger syndrome
- 3. Irreversible vascular obstructive lesion

71. Causes of hematuria in IE:

- 1. Renal infarction
- 2. Glomerulonephritis

72. HF occur early in:

- 1. AV canal
- 2. TGA

73. Drug induced anemia:

- 1. Aplastic → Chloramphenicol, sulfa, cytotoxic drugs
- 2. Immunologic → Penicillin & methyl-dopa

74. Prevention in:

- 1. RF
- 2. IE
- 3. Fe deficiency anemia
- 4. Hemophilia

75. Protected against malaria:

- 1. SC trait
- 2. G6PD deficiency

76. Hyperglycemia causes:

- 1. VOC in SCA (hyperosmolar plasma absorb water from RBCs, decreasing amount of dissolved oxygen in RBCs causing sickling)
- 2. ↓ hemolysis in spherocytosis (hyperosmolar plasma absorbs water from fragile spherical RBCs)

77. Causes of Favism + normal G6PD:

- 1. Didn't wait 2 wks before measuring G6PD activity
- 2. NADPH deficiency
- 3. Glutathione deficiency
- 4. Lab error

78. Nodules:

- 1. Aschoff \rightarrow Rheumatic Fever induced carditis
- 2. SC \rightarrow Rhuematic fever SC affection in 2-10%
- 3. Osler \rightarrow IE minor criteria (due to immune complex deposition on the bulb of the finger)

79. Incidence of IE:

- 1. Lt > Rt \rightarrow since P is higher on the Lt, therefore, more endothelial injury
- 2. MR > MS \rightarrow Since P gradient across the valve during systole (120-2=118 mmHg) ,which the time during which MR occur, is higher than that during diastole (6-0=6mmHg), which the time during which MS plays its role.
- 3. AS > AR → Since ventricles contraction (120mmHg) is more powerful than aortic recoil (80 mmHg)

Most common pediatric syndromes:

	Syndromes
Down	Trisomy 21
Edward	Trisomy 18
Patau	Trisomy 13
Fanconi	
Crouzon's syndrome	Craniostenosis + exophthalmos
Turner	Monosomy X
Klinefelter	XXY
Morquio	Mucopolysaccharidosis IV
Hurler	Mucopolysaccharidosis I (Severest form of MPS)
Hunter	Mucopolysaccharidosis II
Laron	IGF-1 deficiency → Pathological short stature
Silver Russel	Unipaternal disomy of chromosome 7 → Pathological short stature
Sotos	Cerebral giagantism (due to Gliosis)
Marfan	Connective tissue genetic defect
Ehlers Danlos	Connective tissue genetic defect
Cushing	↑ adrenal cortisol
Nutritional recovery	With initiation of kwashiorkor ttt, the liver enlarges more in the first few days (due to storage of lipids)
Reye	Acute encephalitis with fatty infiltration of the liver due to salicylate administration with viral infection.
Mikulicz	Bilateral parotid & lacrimal swelling with dry mouth
Fragile X	Structural chromosomal abnormality in males producing MR + abnormal features + large testes
Hand & foot	Coxachie A virus infection
Hepatorenal	Renal failure secondary to liver cell failure (due to edema induced hypovolemia → ↓ RBF)
Crigler Najjar type I (AR)	Congenitally absent glucuronyl transferase enzyme
Crigler Najjar type II (AD)	Congenitally deficient glucuronyl transferase enzyme
Bronzed baby	The result of phototherapy in cholestasis
Respiratory distress (RDS)	Surfactant deficiency
Meconium aspiration	IU hypoxia → meconium passes to amniotic fluid → Baby breath meconium if respiration is stimulated while still in utero (as in breech delivery) or still covered by amniotic fluid

	after birth	
Congenital Rubella	Transplacental Passage of Rubella infection in	
	early pregnancy from the mother to the fetus	
Eisenmenger	Reversal of Lt to Rt shunt in congenital	
	cardiac defects	
Schwachman-Diamond	Inherited pure red cell aplasia (AR)	
TAR	Thrombocytopenia with absent radius	
Giant platelet	Adnormal platelets causing non-	
	thrombocytopenic purpura	
Kasabach-Merritt	Hemangioma → thrombocytopenia	
Evan	Thrombocytopenia + anemia (Rare)	
Bernard-Soulier	Glycoprotein 1b deficiency →Platelet	
	dysfunction (=Hemorrhagiparous	
	thrombocytic dystrophy)	
Glanzmann's-Naegeli (thrombasthenia)	Glycoprotein IIb/IIIa deficiency →Platelet	
	dysfunction	
Sudden infant death	A complication of GERD	
Sandifer's	Dystonic movement of neck (A complication of GERD)	
Alagille	Paucity of intrahepatic bile ducts + Cong.	
	Heart & vertebral anomalies + Corneal	
	anomalies & triangular face	
Alport	Familial nephritis + deafness +lenticonus	
Nephrotic	Edema + proteinuria + hypoproteinemia &	
	hyperlipidemia	
Nephritic	Glomerulonephritis	
Prader-Willi	Deletion in chromosome 15	
Sturge Weber	Neuro-cutaneous disorder characterized by MR	
Guillain Barré	Symmetric ascending paralysis	
Oral allergy	Fruit allergy → itchy mouth	

Most common cause of:

Most common Cause of	
Proportionate short stature	Normal variant (90%)
Acute laryngitis/ larngotracheobronchitis	Parainfluenza virus (75%)
Septicemia	Meningococci
Death in hospitalized DKA patients	Brain edema
Neonatal mortality	Preterm
Neonatal seizures	HIE
Pathological indirect hyperbilirubinemia	Hemolytic disease of the newborn (Rh & ABO incompatibility)
Neonatal respiratory distress in preterm	RDS (Surfactant deficiency)

Neonatal respiratory distress in full term	Transient tachypnea of newborn	
Neonatal respiratory distress in Post term	Meconium aspiration	
Foetal macrosomia	Maternal diabetes	
Bleeding in healthy new-born	Hemorrhagic disease of the newborn (transient deficiency of vit.K dependent coagulation factors)	
Early onset neonatal sepsis	-Gram -ve enteric bacilli: Klebsiella & E. coli -Enterococci -GBS	
Late onset neonatal sepsis	-Coagulase -ve staphMRSA -Gram -ve enteric bacilli -GBS	
Neonatal sepsis in Egypt	Gram -ve bacilli: Klebsiella, Pseudomonas & E. coli	
Congenital heart disease	Polygenic inheritance	
IE	Gram +ve bacteria: α-hemolytic streptococci, staph. Aureus & coagulase -ve Staph.	
Anaemia in infancy	Iron deficiency	
Hemolysis	G6PD deficiency	
Purpura	ITP	
Acquired bleeding tendency	ITP	
Inherited bleeding tendency	Von-Willebrand disease	
Acute nasopharyngitis	Viral → Rhinovirus	
AOM	Bacterial → Pneumococci, H.influenza, staph., B.catarrhalis, S.pyogenes	
Acute cough in children	Acute bronchitis	
Acute bronchitis	Viral → Adenovirus & parainfluenza virus	
Respiratory distress & wheezes in infancy	Acute bronchiolitis	
Wheezing in children	Bronchial asthma	
Acute bronchiolitis	RSV (80%)	
Pneumonia	RSV	
Bacterial pneumonia below 6 years	Pneumococci	
Bacterial pneumonia above 6 years	Mycoplasma	
Pulmonary TB	Mycobacterium tuberculosis	
Recurrent abdominal pain	Dysfunctional (= non-specific = psychogenic) (>90%)	
Chronic abdominal mass	hepatosplenomegaly	
Intestinal obstruction between 3 month & 3 years	Intussusception	
Diarrhea in infants & children	Infective diarrhea (gastroenteritis)	
Bacterial gastroenteritis	Campylobacter jujeni (E. coli comes in the 2 nd place)	
Viral gastroenteritis	Enteroviruses (HAV, Polio, Coxsackie & Echo)	

Cause of chronic hepatitis in developed countries	NASH (Non-alcoholic steatohepatitis)
Neonatal cholestasis	Idiopathic neonatal hepatitis (Giant cell hepatitis) [EHBA is 2 nd most common]
Nephrotic syndrome	Idiopathic (90%)
UTI	E. coli (90%)
Chronic kidney disease in infancy	Developmental (Renal aplasia, hypoplasia or dysplasia)
Epilepsy	Idiopathic (80%)
Non-epileptic convulsions	Febrile
Encephalitis	Herpes simplex
Congenital obstructive hydrocephalus	Aqueduct stenosis
Floppy infant	Werdnig Hoffmann disease
Acute paralysis in children	Guillain Barré syndrome
Progressive motor weakness	Duchenne muscular dystrophy
1 ^{ry} hypothyroidism	Complete absence of thyroid gland (aplasia or agenesis)
Delayed puberty	Constitutional
True precocious puberty	Idiopathic (80% in female & 50% in male)

Most common:

Most common		
Form of malnutrition	Nutritional dwarfism	
Type of rickets	Vitamin -D deficiency rickets	
Infection before vaccination	Measles	
Food allergens	Milk, eggs, wheat & soy (MEWS)	
Neonatal birth bone injury	Fracture of the clavicle	
Neonatal birth intra-abdominal visceral injury	Liver hematoma	
Choanal atresia	Bony	
Type of congenital heart disease	Non-cyanotic (80%)	
Type on congenital Non-cyanotic heart disease	VSD (30%)	
Type of congenital cyanotic heart disease	Fallot's tetralogy (5%)	
Type of defect in ASD	Ostium secundum (80%)	
Cardiac lesion in Down syndrome	Endocardial cushion defect = Atrioventricular septal defect = ostium primum defect	
Type of aortic stenosis	Valvular (deformed or bicuspid aortic valve)	
Acquired heart disease in children	Rheumatic fever	
Rheumatic heart disease	Mitral regurge (While, Mitral stenosis is most rare in children since it needs 5-10 years)	
Chronic hemolytic anemia in Egypt	B-Thalassemia (AR)	
RBC enzymopathy	G6PD deficiency	

Type of acquired aplastic anemia	Idiopathic (70%)
Type of congenital aplastic anemia	Fanconi anemia
Form of childhood malignancies	Acute Leukemia
Type of acute leukemia in children	Acute lymphoblast leukemia (ALL) (75%)
Type of ITP	Acute (85-90%)
Infection in children	Acute nasopharyngitis
LN affected by tuberculous lymphadenitis	Cervical LNs
Affected vertebrae by Pott's disease	Lower thoracic (as it lies beside the costophrenic space containing exudate full of TB bacilli)
Site of kyphosis in Pott's disease	Mid-thoracic
Pediatric complaint	Abdominal pain
Pancreatic mass due to blunt trauma	Pancreatic pseudo cyst
Site of intussusception	Ileocecal
Form of acute hepatitis in children	Icteric hepatitis
Form of acute hepatitis in infants	Anicteric hepatitis
Presentation of portal HTN	Hematemesis (& earliest manifestation)
Type of primary nephrotic syndrome	Minimal change (75%)
Form of enuresis	Nocturnal
Type of CP	Spastic (70%)
Muscular dystrophy	Duchenne
Food allergy in infants	Milk, egg & peanut
Food allergy in children	Peanut, tree nut & fish
Affected joint in septic arthritis	Knee
Type of JIA	Pauciarticular type 1

TABLES

1. Marasmus vs Kwashiorkor:

	Marasmus	Kwashiorkor
Onset	Chronic	Subacute
Туре	Undernutrition	Malnutrition
Cause	↓ milk / formula	
	concentration or amount	
Edema	Absent	Present
Psychological	Irritable & anxious	Apathy

Marasmic ketosis → Associated with Hypoglycemia
DKA ketosis → Associated with Hyperglycemia

2. Fallot vs TGA:

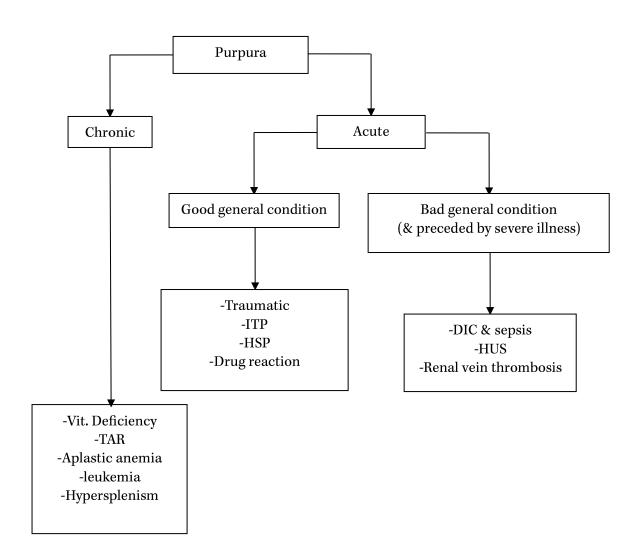
	Fallot	TGA
Time of presentation	1-3 months	At birth
Clubbing	1-2 y	Before 1 y
Cardiomegaly	Absent	Present
Auscultation	Ejection systolic murmur	No murmur
S2	Single	
Chest infection	Absent	Present
HF	Absent	Present
X-ray	Boot-shaped heart	Egg on side shaped heart
Timing of total correction surgery	At 6 months	First 3 wks

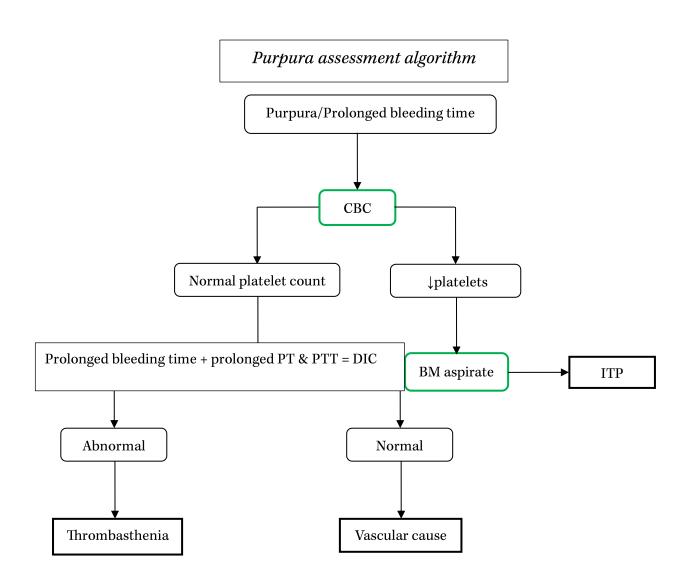
3. Bleeding disorders:

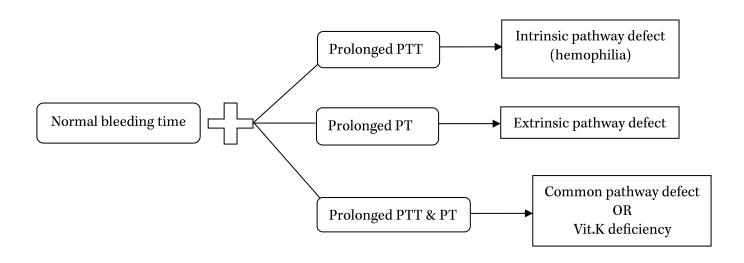
	ITP	Hemophilia	HSP	DIC
Platelet count	↓	Normal	Normal	$\downarrow\downarrow$
Bleeding time	↑	Normal	Normal	↑ ↑
PTT	Normal	↑	Normal	$\uparrow \uparrow$
PT	Normal	Normal	Normal	$\uparrow \uparrow$

DIAGRAMS & ALGORITHMS

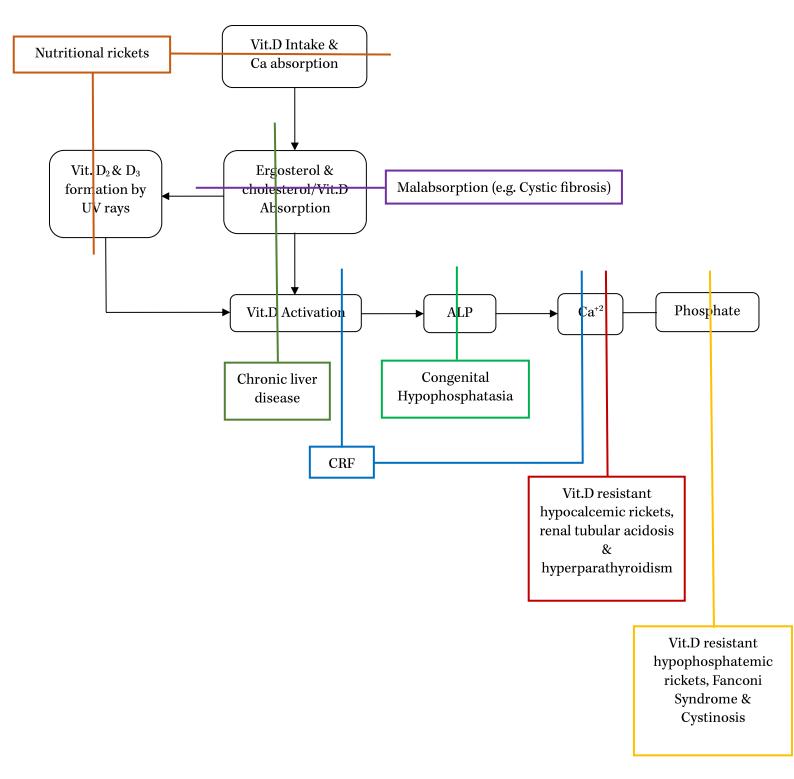
Purpura classification







Rickets etiology diagram



N.B. "This diagram represents the interruption of the normal physiological events by Nutritional & non-nutritional rickets causes"

First sign to appear in rickets → Craniotabes (behind the ear)
Earliest manifestation of rickets: High serum ALP (last to normalize)

KEYWORDS

Infection

Disease	Investigation (Key words)	TTT
Mumps Parotitis Pancreatitis Encephalitis	 Painful swelling in-front & behind the ears with no improvement by analgesics On pressure on swelling → white discharge abdominal pain & vomiting Headache - convulsions - vomiting 	AnalgesicsAntipyretics+ ttt of complications
Scarlet fever	Fever, sore throat, abdominal pain Fever, nausea more with maculo- papular rash	 Penicillin V. (oral) Procaine penicillin Erythromycin Antipyretics Follow up for detect of complications
Roseola Infantum	High fever without clear cause for 4 days then subside with spready rash over the trunk & abdomen + Febrile convulsions	- Antipyretics - Sedative
Rubella	Low grade fever for 1 day diffuse maculopapular rash + Tender occipital lymphadenopathy	- Protection (no specific ttt)
Measles	Fever for 3 days + maculopapular rash on next 3 days With kolpik's spots	Vitamin AAntipyreticsAntibiotics
Herpes simplex infection	 Fever Cervical adenopathy foul breath painful oral lesion on his tongue, gums & lips kerato-conjunctivitis 	
Tetanus of neonates	Generalized tonic convulsions, Failure to suckle, dirty infection of umbilical stump	PenicillinIgTetanus antitoxin
Meningocele septicemia	High feverdrowsinessVomiting	

	- blue mottling patches with respiratory distress	
Impetigo	Non-painful rash then papule then pustule then coalesced in honeycomb like crusts	
Infectious mononucleosis	 Fever marked enlarged cervical & inguinal lymphadenopathy + splenomegaly 	
Chicken pox	 Fever running nose rash popular pustules crustations without scar 	- Prophylaxis - + 4A A

Neonatology

Disease	Investigation (Key words)	TTT
SGA (IUGR)	Perinatal asphyxia Pregnancy induced HTN Infant of toxemic mother	
IDM	 Polycythemia Jaundice Birth injury Macrosomia Hypoglycemia Hypocalcemia RDS & TTN VSD Seizures 	- Monitor: Glucose Hb Bilirubin Electrolytes - Compensate any abnormality
ABO	Mother → O	
Incompatibility	Baby → A, B, AB	
Rh incompatibility	Mother → negative Baby → positive	 Induction of labor at 33 weeks Blood exchange Human anti-D globulin
Meconium aspiration syndrome	 Mother → DM, HTN, Prolonged labor Baby → post-term & fetal hypoxia Symptoms of RDS Hyperinflated lung X-ray → streaky areas of collapsed lung 	- Suction - Oxygen & mechanical resuscitation
Choanal atresia	Cyanosis during feedingWhich relieve by cryingCatheter into nose not pass	Surgical correctionNasal or oral nasolaryngeal tube
Diaphragmatic hernia	Scaphoid abdomenNo breath found on left side of thoraxNo improvement with ETT	- All solvings are accepted except IPPV or CPAP
RDS	 Preterm < 37 months - CS Tachypnea (grade I) >60\minutes Intercostal and subcostal retraction (grade II) Cyanosis (grade IV) Grunting (grade III) Chest X-ray → white lung 	 Decreased lung compliance Decreased lung volume Embryo circulation Right to left shunt

Hubocalonnia	Forly.	- < 6.5:
Hypocalcemia	- Early: 1- Preterm infant	- < 6.5: 5 ml\kg\24 10%Cugh
	2- IDM	- In apnea or failure:
	3- IUGR asphyxia	2 ml\kg\ of 10% of calcium
	- Late:	gluconate in 5 minutes
	1- Increased phosphate level	
	2- Immature parathyroid	
Convulsions	- Hypoglycemia <40	
	- Or hypocalcemia <7	
	- Or hypoxia of the brain	
	- Magnesium	
	- Preterm of DM	
	- + or – sodium	
	- HIE	
Hypothermia	- Temperature <35 c → warmer	
TTN	- Full-term + CS +distress – cyanosis	
	+ pulmonary vascular markings	
STORCH	- Petechiae in new born + seizures	
(congenital	- Thrombocytopenia	
infections)	- Hepatosplenomegaly	
Sepsis	- Fever, bulging anterior fontanel, no	
	neck rigidity, feeding problem	
	الأم هتقولك الولد سخن وهمدان ومش بيرضع	
	ومصفر وعنده اسهال وترجيع وبطنه منفوخة	
	وبيتشنج	
Heart failure	3T:	
	- Tachypnea	
	- Tachycardia	
	- Tender liver	
RDS	- بیسأل عن: grades, X-ray	
120		
	- Tachypnea	
	- Intercostal and subcostal retraction	
	- Expiratory Grunting	
	- Chest X-ray → reticular granular	
Hemorrhagic	- Bleeding with normal platelets count	
disease of new	"Vitamin K deficiency"	
born		
Birth injury	- Pallor with occult bleeding "intra-	
 .	abdominal, intra-cranial"	
	- Cephalohematoma	
	Subgaleal hematoma	
	IC hematoma	
	caput succedaneum	
	- Due to prolonged labor	
	- Due to brototiged tapor	

Jaundice	- Total bilirubin >15	
	- Direct bilirubin: 1 or 2	
	- Enlarged liver → biliary atresia	
	- First day → ABO – Rh incompatibility	
	- Second & third day → physiological	
	case	
	- Fourth day → breast milk jaundice & hypothyroidism	
Anemia	- Pallor in color	- Erythropoietin in preterm
	- Birth injury (Cephalohematoma,	(prophylactic)
	Subgaleal, caput succedaneum, IC hemorrhage)	- RBCs exchange – Whole blood
	- Occult bleeding (IC hemorrhage, intra-abdominal hge	
	- Occult blood loss (feto-maternal	
	transfusion, feto-fetal transfusion)	
	- Occult blood loss come in case after exclusion of other reasons	
Polycythemia	- LGA (IDM)	
	- Hypoxia:	
	* Infant of toxemic mother → HTN	
	* placental aging → post-term	
	newborn	

Cardiology

Disease	Investigation (Key words)	TTT
ASD	 May be normal check-up Fixed wide split S2 Ejection systolic murmur on pulmonary area 	- Main ttt closure: *catheter *surgical - Medical ttt for complication
VSD	 Symptoms of heart defect: Shorten of breath Difficulty in feeding Tachypnea – Irritation Pan-systolic murmur over left lower sternal border 	 Main ttt closure: *catheter *surgical Medical ttt for complication
PDA	 Continuous machinery murmur Load S2 	 1st weak → Indomethacin 1st year ends → closure: *catheter *surgical
Teratology of Fallot	 Cyanosis on feeding Systolic ejection murmur on pulmonary area Chest x-ray→ mild Right ventricular enlargement & small pulmonary artery Clubbing of fingers 	 Prostaglandins infusion Deeply cyanosed neonate → Palliative shunt Total correction at 6 months with patch closure
TGA	 Single S2 Cyanosis since birth which exaggerates in second day X-ray → egg-shaped heart Clubbing of fingers 	 Prostaglandins infusion Rashkind septostomy (first day) Total correction in first 3 weeks of life
Digitalis toxicity	VomitingCardiac arrhythmiaVisual disturbance	• Fab
Rheumatic fever	 Major → ACCES Minor → AAEEFF Evidence → throat culture & Antistreptococcal antibodies Investigations: evidence for streptococcal infection ASOT, throat culture Degree of inflammation: CBC, ESR, CRP 	 TTT → Aspirin & steroid TTT of chorea TTT of complication Primary prevention Secondary prevention
Heart failure	3T:	- Diuretics

	- Tachypnea	- Digitalis
	- Tachypnea	- Oxygen therapy & fluid restriction
	- Tender enlargement	- First action in the case
	+ Communication of NCD ACD	anti-failure ttt and VD in
	- Symptoms of VSD, ASD	grade III heart failure
		- After management → ttt
		of the complication
Septic arthritis	- Inability to move, swollen, tender knee	- AB ttt → local and
	joint	symptomatic
	- Most common organisms are H.	- Drainage
	Influenza and staph. Auris	- Surgical connection
	- Joint fluid examination (Tapping)	
Coarctation of	- Radio-femoral delay	- Stent (من جوة)
aorta	- Cyanosis on exercise	- Resection (BV)
	- Systolic murmur over the back	- Graft (من برة)
	accentuated S2	
	- Differential cyanosis	
Infective	- Fever > 38C – splenomegaly	- Penicillin – gentamycin
endocarditis	- Vascular or other immune signs	- Surgical with persistent
	- Signs of Carditis and congenital heart	bacteremia and
	- Heart failure	progressive heart failure
Carditis	- Tachycardia	- TTT of rheumatic fever
myocarditis	- Tachypnea	
Rheumatic fever	- Or Dyspnea	
	- Muffled heart sounds	
Cardiogenic	- Low BP	- Dopamine → first line
shock	- High heart rate or pulse	- Preload ttt
	- Enlarged liver	- Afterload ttt
	- Increased capillary refill	- Arrhythmia ttt
Aortic stenosis	- Murmur of aortic area	- If pressure gradient > 50
	- Ejection systolic click	mm HG:
	- Dyspnea on exercise	Balloon valvoplasty
	- Echocardiography → aortic stenosis	 Valvotomy
		Valve replacement
Pulmonary	- Load harsh murmur on pulmonary area	- Balloon valvoplasty
stenosis	without any signs for congenital heart	(من جوة)
		- Valvotomy (BV)
		- Valve replacement (من برة)

Hematology

Disease	Investigations -keywords	Management
IDA	 History: prolong Breast Feeding - cow milk -Picky eater dyspnea - tachycardia CBC - ↓HB - microcyte hypochromic blood chemistry ↓iron ↓ferritin ↓TIBC 	1. 1 st line \oral iron 2. 2 nd line \ IM iron for 2months or 4-8 weeks after normalization CBC
B-thalassemia major	 History –consanguinity – 2nd 6 months of life CBC – microcyte hypochromic anemia target cells fetal Hb *A2 HB in B-thalassemia trait 	SS+ RBCs 1- supportive 2- packed RBCs 3- chelating agent 4- splenectomy
Sickle	 History any vaso-occlusive crisis CBC sickle cells 20-40 % HS trait >90 % HS major any investigation for crisis is chemistry of blood 	1- O2 and fluid transfusion 2- blood transfusion 3- exchange transfusion 4- bicarbonate 5- complete bed rest ABBCG
G6PD	 Neonatal jaundice analgesic palpitation antibiotic dyspnea antimalaria drowsiness Acute hemolysis normochromic normocytic jaundice hemoglobinemia hemoglobinuria 	Urgent packed RBCs transfusion chelating if repeated
H-spherocytosis – hereditary الآباء مش لازم یکونوا مصابین	25% mutation (AD) parents not have the dis	RBCs transfusion chelating splenectomy
Any case occlusive	CBC	1st IV fluid & o2

crisis		2 nd bl. Transfusion
		3 rd exchange transfusion
Aplastic anemia	- Purpura	BMT
	- pale in color	immunosuppressive
	- ecchymosis & bleeding	ATA
	- ↓blood para meter	
Acute leukemia	- Most common (ALL)	Supportive ttt
	- bone pain	chemotherapy
	- Aplastic anemia	
	- lymphadenopathy	
	- hepatosplenomegaly	
Fanconi	- Aplastic anemia	1) 1 st Androgen
	- absent radius	corticosteroid
	- nephritis	2) BMT
	- Microcephaly	
	- Microphthalmia	
	- mental retardation	
	- ↓body proportion + pigmentation	
TAR	Thrombocytopenia only with absent	
	radius	
Purpura	- In lower limb →HSP	
	with splenomegaly → leukemia	
	- (purpura on mucous membrane) generalized	
	- purpura →ITP	
	- epistaxis →VWD	
	- blood collection → hemophilia A	
ITP	- Acute 40% chronic 10 %	Prednisone
	not blanching – not raised	IVIG → acute then
	- red turns to green within 2 days	plasmapheresis
	- (purpura on mucous membrane)	splenectomy chronic
HSP	- Lower limb purpura	Salicylate
	- arthritis joint of LL	steroid
	- intussusption GIT	
	- nephritis	
	- Testicular hemorrhage	
		- I

Respiration

Disease	Investigation Key words	TTT
Lower respiratory tract infection	Prodroma: upper respiratory tract infection for 2 daysThen cough, dyspnea	
Acute bronchiolitis	 Cough+ runny nose sneezing couple Expiratory wheezes Respiratory distress (Tachypnea) Chest hyper expansion Chest x-ray: "hyper inflated chest" 	 Humidified O₂ + IV fluid Hand hygiene High frequency MV 3H Caused by RSV
Acute bronchitis	 Prodrome for 2 days of common cold "sneezing" "running nose" then Low grade fever-cough No respiratory distress No wheezes No chest signs in x-ray May come late with high fever In 2ry bacterial infection 	 Most cases recover spontaneously Expectorants and mucolytics Antipyretics Antibiotics
Whooping cough	Paroxysmal stage of attacks if spasmodic cough followed by vomiting of large amount of sputum	Erythromycin + symptomatic & complication ttt
Acute otitis media	 Fever-ear discharge if perforated drum Bright red bulging ear drum Continuous crying and ear nabbing 	Broad spectrum AbsAnalgesics antipyretics
Nasopharyngitis Common cold	Mild feverrunny nosemalaiseVomitingHyperaemic throat	Paracetamol only as antipyretics

Disease	Investigation keywords	TTT
Pneumonia	 Fever-cough-difficult breathing-respiratory distress+ referred pain in neck or abdomen RR > 40 bronchial breathing on auscultation on affected lobe Chest x-ray heterogenous opacity CBC, WBCs(high) band cells ++ No wheezes except in interstitial pneumonia "caused by RSV" if patient develop sudden respiratory distress + hypotension +poor perfusion complicated pneumothorax 	 1ST line –supportive ttt O₂ +IV fluid Need hospitalization if sever cases
TB	 TB toxemia Loss of weight -night seat Loss of appetite-night fever TB spread Fever-malabsorption Lymphadenopathy + VIP chest x-ray +ve tuberculin test 	- All anti TB are oral except streptomycin - RIP"1st line " 1- rifampicin oral 2- isoniazid (DOC) oral 3- pyrazinamide oral - 2ed line 1- Streptomycin IM 2- Ethambutol oral 3- Ethionamide oral
Bronchial asthma	 Repeated respiratory distress & cough especially at night The condition has family history of parents or family Night cough not improved by decongestant O₂ saturation decreases Wide spread expiratory wheezes 	1 ST line SABA 2 ND steroid
Diaphragmatic hernia Acute laryngitis	 Respiratory distress No air entry on left side of the chest Mild respiratory distress Stridor Barking cough Para influenza virus 	CI CPAP or MV - Humid O ₂ & Iv fluid - Nebulized epiprophine - Systemic steroid
Eczema	Fever dyspnea -wheezesUrticariaPenicillin infectionMEWS eating	1^{ST} IM or ID adrenaline 2^{ND} steroid
Tonsillitis	Fever-tonsillitis - retropharyngeal abscessTonsillitis - stridor	

	- Peritonsillar abscess	
Complicated pneumothorax	- Pneumonia then sudden development of respiratory distress	1 ST line chest tube
	- Tachypnea	
	- O_2 saturation	
Aspiration	- Gasoline intake with cough	- 1^{ST} line- O_2 therapy
pneumonia	- Tachypnea -sub costal retractions	- ABG
	- Case	- CI
	 Vomiting then stopped 	- Stomach wash
	$ullet$ $2^{ ext{ND}}$ day—respiratory distress	- Induced vomiting
Laryngeoedema	- Noisy breathing	
	- Worsen during supine position	
	- Inspiration stridor	
Foreign body	- Sudden onset of cough-stridor	- Suction
aspiration	- Intercostal retraction	- Bronchoscopy
	respiratory distress (Tachypnea)	- If lung collapsed-chest
	- Chest x-ray localized body in chest (trachea-bronchi)	tube
	- Wheezing	
Choanal atresia	Neonate with respiratory distress which	- Orotracheal tube
	improve on crying	- Oral airway tube
		- Nonlaryngeal tube if unilateral

Disease	Investigation keywords	TTT
Tracheo-	- Wheezes &cough during feeding	Surgical correction
Esophageal fistula	- Since birth	
	- Expiratory wheezes	
	- Investigations: upper gastrointestinal contrast	
	- Or chest & neck x-ray with Ryle tube in oesophagus	
Cystic fibrosis	- Poor weight gain	
	- Persistent cough	
	- Several bouts of pneumothorax	
	- Large amount, foul smelling	
	- Stool for long period	
	- Sweat chloride test	
	- Na ⁺ >60 MEq/dl	
	- Normal is 30 MEq/dl	

GIT

Disease	Investigations and keywords	TTT
Acute intussusception	 - 1st: vomiting& crying. - 2nd: sausage like mass or drawing the leg over the abdomen. - 3rd: RE blood stool or red currant jelly stool. - Investigations: X-ray: multiple air fluid. X-ray with contrast. Abdominal u/s 	Reduction or resection N.B: Intussusception occur mostly in ileocecum so most probable will be under liver mass (upper right quadrant)
CHPS شیبسی بالزیتون	 3-4 weeks of age. Projectile vomiting. (Non projectile vomiting). Olive like mass on Rt side of umbilicus. Vomiting after 2 months Peristalsis movement during feeding 	Fluid correction.Pyloromyotomy.
Dysfunctional abdominal pain.	 Recurrent pain Not tender Around umbilicus. Normal labs. Stool analysis. عشان تطمن الأم 	reassurance
Git malrotation	 Bilious vomiting Pain X-ray with contrast: Show abnormal site for flexure and other parts of git. 	Surgical correlation

Disease	Investigations and keywords	TTT
Congenital megacolon.	2-3 days of age.No stool passing.Abdominal distention, vomiting.	Surgery
Congenital abdominal hyperplasia	Vomiting, dehydration.Skin hyperpigmentation.	Medical. Chemo & radiotherapy. Surgery.
Neuroblastoma. BM → pallor & petechia.	 Below 3 years of age. Abdominal distention. Fever. Pallor+ petechial spots. Liver ptosis& hepatomegaly. Hard mass on left hypochondrium. BM/diagnostic method. 	Once therapy. NB: - Suprarenal gland → cortisol ↑glucose. - aldosterone →Na retain. So, Neuroblastoma→ hyponatremia& hypoglycemia.
Wilms tumor	 - Age≥3 years. - Hematuria. - Abdominal distension. - Masses on Rt& Lt hypochondrium. 	 Onco therapy. Ct abdomen & biopsy → investigation.
Dehydration.	 Skin-MM- orifices of head. Tear-urine-RR- HR. Pulse-general condition. (Detect the degree). 	 shock therapy. deficit therapy. Maintenance therapy KCl solution15%. ORS.

Disease	Keywords & investigations	TTT
Duodenal atresia.	- bilious vomiting.	
	- passing meconium.	
	- gastric distension.	
	- Age below 1 month.	
Celiac disease	- Abdominal distention.	Gluten free diet.
	- Diarrhea.	
	- Body wasting. → during time of weaning by wheat product.	
Familial	Acute recurrent attack of abdominal pain	VIP
Mediterranean	every 1-2 month/ with arthritis	
fever.		
Fecal mass	- Recurrent abdominal pain.	Laxative.
	- Constipation.	
	- At evening before sleeping.	
Hench schonalin purpura	- Acute abdominal pain without control by analgesia.	
ригриги	- Purpura in LL	
GERD	- 30% of children in 1st year.	- Mild: position and thick
	start from 2nd week but child grow	food
	normally with improvement from 1 month of age.	- Moderate: domperidone and omeprazole
	month of age.	- Severe: fundoplication
		bevere, fulldopheadon

Hepatology

Disease	Key word, investigation	TTT
Acute	-Acute abdominal pain in RT upper quadrant	-prevention
hepatitis	-jaundice -pruritis – mild hepatomegaly	-general
	-enlarged tender liver	-vaccines {A, B}
	Swelling-hepatomegaly	-actual ttt
	Hotness→low grade fever	-Antiviral (A, B, C)
	Tenderness→ enlarged tender liver	-interferon
	Redness → jaundice	- {A, E} spontaneous
	SHATR	
	Investigations:	
	C→ anti HC, PCR	
	B →markers	
	 HBsAg-→acute 	
	HBsAb -→vaccine /Recovery	
	 HBcIgG→chronic 	
	-→ A markers	
	{IgM→recent /IgG→recovery and immunity}	
Neonatal	-Jaundice- pruritis- hepatitis then percutaneous	بتظبط العيان عشان تنقل كبد
cholestas is	liver biopsy	1- replacement therapy
	-clay colored stool -bleeding	2- displacement operation
	HIDA SCAN	3- symptomatic
	-fever- nonbilious vomiting	4- transplantation
	Investigations:	
	1- Bilirubin & AlT, AST	
	2- Liver function	
	3- 6 steps investigations	
	4- Treatable	
	5- STORCH screening	
	6- Other metabolite	
portal	postural hypertension	In case of hepatitis
HTN	• Ascites	complicated by portal HTN:
	Splenomegaly or HSM	it's emergency so do not go
	Collateral circulation	for TTT of hepatitis now,
	• hematemesis {NIP}	manage your case for bleeding or ascites or
	Investigations:	splenomegaly first then go
	Vascular disease due to hepatic disease	to hepatitis.
	- VASCULAR INVESTIGATIONS:	Treatment:
	1- Endoscopy	1- Manage bleeding

2- CTA-MRV	a. IVF
3- doppler -U/S	b. Plasma
- HEPATIC INVESTIGATIONS:	c. whole blood
1- liver function	transfusion
2- casual INV	2- Endoscope
3- hepatitis marker	a. Vasopressive
4- Autoimmune markers	b. Sclerotherapy
5- TMS-sweat	c. Band ligation
6- Liver biopsy	3- surgery
	a. TIPS
	b. pss
	PREVENTION
	1 st line
	1- Avoid NSAID
	2- BB
	3- SCLERotherapy
	$2^{ m nd}$ line
	1- BB
	2- Sclerothetherapy
	3- PSS
	4- LT

Neurology

Definition	Cl/P and Investigations	Treatment
Guillain Barre Syndrome EMG	-GIT infection -Inability to walk (Progressive to bulbar paralysis) -Respiratory failure -Absent deep reflexes	-ICU - mech vent Then IVIG and Plasmapheresis -Physiotherapy
Duchenne Muscle Dystrophy EMG	-Difficult in climbing stairs -Waddling gait -Calves muscle enlargement on examination -Lordosis during walking	Gower's Signs - Conservative -Respiratory aids + Steroid
Convulsions	-1st attack or 2nd attack (epilepsy is not diagnosed by first attack, we diagnose it if recurrent, so we deal with first attack by anticonvulsants only, also we use them in epilepsy as emergency management salivatory and gargling noises with inability to speak and no response to verbal order (immbending convulsion so deal as convulsion) -Fever + vomiting + seizures= febrile convulsions. → 5 criteria of febrile: 1- fever 2- vomiting 3- seizures 4- no history for previous convulsions 5- evidence for extra cranial cause not intra cranial cause and family history - EEG	- Anticonvulsant (diazepam) (most appropriate rescue therapy) - Antiepileptics 1. Neonatal (Phenobarbitone) 2. Child or infant (Dipakin) - After management search for cause 1. Vital sign specially BP 2. Neurological examination 3. Cutaneous and abdominal
Head Trauma	-Sudden onset of lateralization signs -Unconsciousness -Hemiplegia	- CT Brain

Cl/P and investigations	Treatment
-IUGR + Purpura + HSM	CSF culture
-Prolonged neonatal jaundice	Blood culture
	Serology
	TORCH screening
-Purpura fulminous (rapid spreading non-	
bleeding rash)	
→ Rapid progressive DIC	
→ Shock	→ So first line of ttt shock
-Fever + cough + poor growth = TB	therapy
+ sign for ICT (increased cranial tension)	→ TB meningitis, take TB
*CSF examination	therapy
-Delayed development	
_	
V 1	
-nespiratory infection	
(from neonatal period till now)	
-Flaccid paralysis	Treatment of pulmonary
(bulbar paralysis)	complications
-Dry pregnancy??	+
* * * *	
	Nasogastric tube
Than ogryposis	
-Worm like fasciculations of tongue	
	-IUGR + Purpura + HSM -Prolonged neonatal jaundice -Purpura fulminous (rapid spreading nonbleeding rash) → Rapid progressive DIC → Shock -Fever + cough + poor growth = TB + sign for ICT (increased cranial tension) *CSF examination -Delayed development -Floppy baby -Hyperreflexia -Respiratory infection (from neonatal period till now) -Flaccid paralysis (bulbar paralysis) -Dry pregnancy?? -Decreased fetal movement -Arthrogryposis

Disease	Cl/p and Investigation	
Poliomyelitis	-Unilateral lower limb motor weakness with normal sensation	
Midline post fossa tumor	-Rapid, progressive increased ICT with ataxia or gait -Instability -Papilledema, headache, projectile vomiting	
Petit mal seizure	Consciousness impairment for 5-20 seconds	

Endocrinology

Disease	c/p and investigations	Treatment
Premature thelarche	Breast enlargement without another organ enlargement	Reassurance
Gynecomastia	Without discharge	Reassurance
Premature adrenarche	Pubic hair development without other 2ry characters	Reassurance
True precocious puberty	2ry sex character appear, enlargement of gonads ovulation or spermatogenesis before 4-8 years	If idiopathic reassurance if you detect the cause treat it
Pseudo precocious puberty	2ry sex character appear without gonads enlargement	Ttt the cause tumor or excess hormones
Delayed puberty	Delayed appearance of 2ry sexual ccc after 8-14 years	FSH –LH testosterone puberty hormone profile exclude systemic diseases
Gynecomastia in male	Breast enlargement with discharge	FSH, LH, serum testosterone + breast U/S
Hypothyroidism	lry ↓T3, T4 ↑TSH 2ry ↓T3, T4↓TSH +CP at birth / screening newborn /constipation Abd distension prolonged jaundice excess sleep childhood /delayed sexual development, motor & mental retarded, protruded tongue	T3 T4, TSH bone age radioactive assay US neonatal screening Treatment: lifelong thyroid hormone continuous monitor of developmental aspects
DM I	Polyuria &polyphagia nocturnal enuresis come with rapid breathing with special odor of breath fasting glucose > 126 come of hypoglycemia due to insulin dose	Lifelong insulin therapy + monitor of development Aspect
DKA	Acute abdomen pain, vomiting history of polyphagia &polydipsia diagnostic criteria →DM →labs	Saline → infusion to correct dehydration , shock ABG

NOTES & MNEMONICS

Neonatology

1. Convulsions (Neonatal seizures):

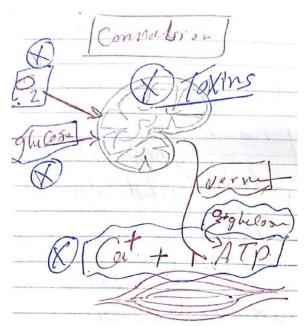
- → Causes:
- A. Hypoxic:
 - 1. Perinatal problem.
 - 2. Polycythemia.
- B. Hypoglycemia:
 - 1. Metabolic problem.
 - 2. \downarrow Ca²⁺ & \downarrow ATP.
- C. Brain toxins:
 - 1. Congenital problems.
 - 2. Infections.
 - 3. Infarction.
 - 4. Drugs.
- D. Unknown.
- **→** *Investigations:*
- A. Lab:
 - 1. Power indicators.
 - 2. Infections & toxin indicators.
- B. Radiology:

أعمل أي أشعة على الخ:

- 1. U/S.
- 2. CT.
- 3. MRI.
- 4. EEG.

→ *Treatment:*

- 1. Phenobarbitone.
- 2. Phenytoin.
- → Prognosis:
 - 1. 15%: Death.
 - 2. 30%: Neurological sequeale.
 - 3. 15-20%: Chronic.
 - 4. 40%: Normal.



2. Preterm:

→ Causes:

Preterm = Immature.

- 1. Mother.
- 2. Uterus + Cervix.
- 3. Placenta + Membranes.
- 4. Fetus.
- \rightarrow Mechanism \rightarrow Hypoxia.
- → Clinical picture:
 - Measures: √.
 - eye, face & skin.
 - breast & genitalia.
 - Lower limb & sole.
- → Complications = causes of death من الشرعى:
 - 1. CNS: أصفر وأحمر وأبيض.
 - 2. CVS: sepsis, retina, metabolic.
 - 3. Respiratory: AAB (Apnea, Aspiration & Broncho-pulmonary dysplasia) + RDS.
 - 4. GIT: NE, GERD & poor suckling.

3. SGA:

→ Causes:

علاقته وحشة مع أمه:

- A. Maternal diseases.
- B. Baby abnormality.
- C. Placenta:
 - 1. HTN.
 - 2. Collagen vascular disease.
 - 3. Multiple gestations.
 - 4. Vascular malformations.
 - 5. Infarctions.
- → Clinical picture:

Compare with preterm.

- A. Measures: \checkmark .
 - 1. Eye, face& skin: mature.
 - 2. Breast & genitalia: mature.
 - 3. LL & sole: mature.
- B. + Umbilical cord: thin.

دي زيادة عن الpreterm.

C. -- Subcutaneous fat.

ودى كمان زيادة.

- **→** *Complications:*
 - 1. Infections (TORCH).
 - 2. Congenital anomalies.

- 3. Respiratory: \downarrow .
- 4. Metabolic: ↓.

4. Hypoxic ischemic encephalopathy:

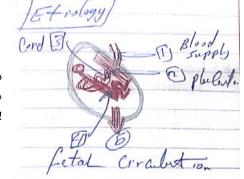
→ *Etiology:*

- 1. Impaired placental flow.
- 2. \downarrow Placental gas exchange.
- 3. Impaired cord blood flow.
- 4. Fetal anemia.
- 5. Birth asphyxia.

→ Clinical picture:

من الواضح إن خلايا المخ هتموت وهو لسه جنين. وبالتالي اC/PJ بتاعته هتيجي وهو جنين. وبالتالي محتاجين أجهزة وتحاليل عشان نعرفها. ولما ينزل من أمه هيكون واضح كل حاجة.

- 1. CTG.
- 2. Cord ABG (fetal).
- 3. At birth: ↓ Apgar score.
- 4. Within 2 days: asphyxia.



	Mild	Moderate	Severe
Feeding	Impaired	Can't	X
Reflexes	Excessive	Abnormal	X
Seizures	X	±	Refractory
Movement	Normal	Abnormal	No movement

→ *Investigations:*

- 1. Prenatal: biophysical profile/US.
- 2. Perinatal: ABG/Apgar.
- 3. Postnatal: Lab/Radiology.

→ Treatment:

- 1. Anticonvulsants.
- 2. Hypothermia ttt.
- 3. MOSF ttt.

5. Jaundice:

→ Causes:

A. Physiological:

Premature: RBCs, liver, GIT.

- B. Pathological: indirect
 - 1. $\uparrow RBCs$ destruction:
 - a. 1st day: ABO-RH, G6PD.
 - b. Sepsis, TORCH.
 - c. Extravasated blood.
 - d. Polycythemia.
 - 2. Defect in detoxification processes:

Liver: Y-Z protein, conjugation enzymes, EHC.

Rh:

- Mother Rh -ve.
- Baby Rh +ve.

ABO:

- Mother: O.
- Baby: A, B, AB.
- → Treatment:
 - 1. Induction of labor.
 - 2. Blood: exchange, transfusion.
 - 3. Anti D, A, B globulin.
- → *Jaundice assessment:*

Time, History, Examination & Investigations.

- 1. Time:
 - 1st: ABO, Rh.
 - 2nd or 3rd: physiological jaundice, polycythemia.
 - 4th 7th: sepsis, Criggler-Najjar.
 - 2 weeks: pathological: enzyme, hormones, cholestasis, hypothyroidism.
- 2. Examination:
 - 1. Skin color: yellow, pale.
 - 2. Size: microcephaly, omphalitis, hepatomegaly.
 - 3. CNS: lethargy, poor feeding, kernicterus.
- 3. Investigations:
- A. Prove jaundice:

Bilirubin (total, indirect).

- B. Prove the cause:
 - 1. Infection \rightarrow hemolysis:
 - a. CBC: Reticulocytosis.
 - b. WBCs, ESR & CRP.
 - 2. Hemolytic disease:

ABO, Rh, Coomb's test.

- 3. Hemolytic anemia:
 - a. Osmotic fragility test.
 - b. Enzyme assay.
- 4. $T_3 & T_4$.
- 4. History:

سؤال للعائلة. سؤال للطفل الأكبر. سؤالين في الحمل. سؤال في الولادة. سؤال في الرضاعة.

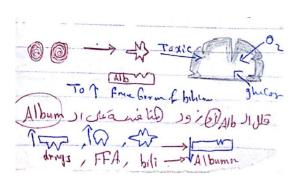
→ *Treatment:*

- 1. Monitor.
- 2. Prevention.

- 3. Phototherapy.
- 4. Exchange transfusion.
- 5. Pharmacologic therapy.
- 6. Treatment of the cause.

6. BIND:

- → Risk factors:
 - ↑ hemolysis → ↑ bilirubin (duration, conc. & free form) + \downarrow brain glucose & O₂.
- → Causes:



→ Clinical picture:

A. Acute:

Early, intermediate,

advanced

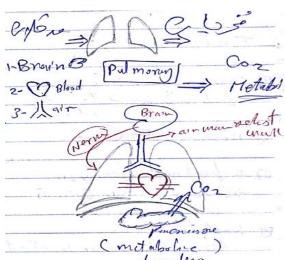
- (according to symptoms not time). • Early: ↓ activity & ↑ cry.
 - Intermediate: ↑ Hypertonia + ↑ Mortality + ↑ Encephalopathy.
 - B. Advanced: نهایته سودا
 - Opisthotonus, cry, apnea, seizures, coma & death.
- → *Chronic kernicterus:*
 - 1. Deafness صمّ.
 - 2. Athetosis بكمٌ.
 - 3. Limitation of gaze عمىّ.
 - 4. Mental retardation فهم لا يعقلون.
 - 5. + Dental dysplasia.
- **→** *Treatment:*

Phototherapy + Blood exchange + Monitoring.

7. RDS:

- → Causes:
 - 1. Pulmonary.
 - 2. Extra-pulmonary.
- A. Pulmonary causes:
 - 3 diseases don't open lung.
 - 3 diseases don't close lung.
 - 2 diseases destruct lung.
 - 3 diseases destruct lung from outside.

B. Extra-pulmonary causes:



→ Factors that increase risk 1st: Premature.

أم عندها السكر حامل في ٢، واحد مات والتاني ناقص، والدكتور ولدها قيصري.

- → Factors that decrease risk of RDS:
 - 1. HTN mother.
 - 2. Premature rupture of membranes.
 - 3. Steroids & T₄.

أم الضغط على عليها، فرقع الmembranes وزود الsteroids.

of RDS:

→ Clinical picture:

سهلة.

→ *Complications.*

Hemorrhage, Air & Fibrosis.

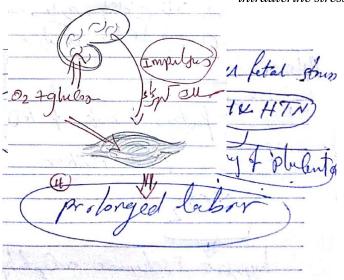
- = Hemorrhage, barotrauma & chronic complications.
- **→** *Treatment:*

Monitor + symptomatic ttt + specific ttt.

- A. Monitor:
 - 1. Vitals.
 - 2. O_2 .
 - 3. Gases.
 - 4. Blood.
 - 5. Parameters.
- B. Symptomatic ttt:

Correction of hypoxemia.

C. Specific ttt + life support.

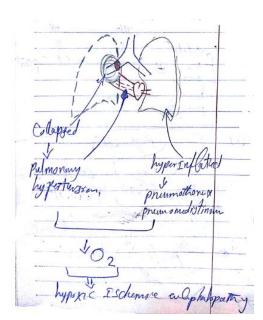


8. Meconium aspiration syndrome.

→ Clinical picture:

As RDS.

- 1. Inspection: grade I, II, III or IV.
- 2. Auscultation: \downarrow breath sounds.
- 3. Stained skin by meconium.
- **→** *Complications:*



Apnea:

Stopping of respiratory movements

> 20 seconds.

Movement = Neuromuscular junction. So Causes:

- 1. Premature brain or muscles.
- 2. Defect in brain or muscles or nerves.

So:

So, 2ry causes:

- Metabolic (↓ glucose).
- Hypoxic (↓ oxygen).
- Toxic (damage).
- اقطع سلوك الكهرباء

Metabolic:

- Hypoxic: hypoxia, seizures, temperature.
- Toxic: hemorrhage, sepsis, drugs.

اقطع سلوك الكهرباء:

- Gastro-esophageal reflux.
- With ETT or suction.
- **→** *Treatment:*

Monitor + symptomatic ttt + specific ttt.

- 1. Monitor: HR, RR.
- 2. Symptomatic: pharmacologic, CPAP, IMV.
- 3. Specific ttt of the cause.

9. Hypoglycemia:

→ Causes:

Glucose + insulin \rightarrow ATP.

- → For hypoglycemia:
 - ↓ glucose sources.
 - ↑ insulin.
 - \uparrow consumption or demand.
- A. \downarrow glucose sources:
 - IUGR.
 - ↓uptake.
 - Metabolic errors.
- B. \uparrow insulin:
 - IDM.
 - Islet cell hyperplasia.
- C. \uparrow consumption:
 - Serious illness: sepsis, shock, asphyxia.
 - RDS, hypothermia, polycythemia.

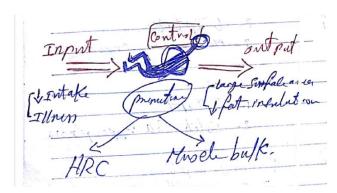
10. Hypothermia.

 \downarrow Insulation.

↑ Auto-consumption.

- A. \downarrow Insulation:
 - Cold environment.
 - \downarrow drying at birth.
 - \downarrow clothing.
- B. \uparrow Auto-consumption:
 - Sepsis.
 - Prematurity.
- → Clinical picture:

Hypothermia: VC (peripheral & pulmonary)/↓ CNS/↓ BMR.



كانوا زمان في عمليات المخ والأعصاب بينزلوا درجة حرارة المخ 5 – عشان يعرفوا يشتغلوا.

11. Infants of diabetic mothers (IDM).

Intrauterine.

 $DM \rightarrow CS$.

↑ Glucose: macrosomia, birth injury or abortion, congenital heart diseases.

↑ Insulin: polycythemia → jaundice, hypocalcaemia & magnesemia.

Macrosomia:

CS.

RDS, TTN.

↑ Insulin:

Hypoglycemia.

Hypocalcaemia & hypomagnesemia.

CNS

In general: diabetic mothers \rightarrow aging of placenta.

So, DM \rightarrow IUGR.

12. Neonatal sepsis.

\rightarrow Risk factors:

- A. Neonate:
 - \downarrow weight (2).
 - \downarrow intake (2).
 - Male.
- B. Mother:
 - Sepsis: fever & leukocytosis.
 - Local infection (2).
 - Hidden infection (2).
- C. Personnel & equipment.

→ Clinical picture:

Not doing well + Any hypo or hyper:

- Glycemia.
- Thermia.
- Pnea.
- Cardia.
- Intake (vomiting, distension).
- + Jaundice.
- + Complications: EP, DIC, shock, bulging anterior fontanel.
- **→** *Investigations:*
- 1. Leukocytosis, neutrophils, immature neutrophils.
- 2. CRP.
- 3. Blood culture.
- 4. Other cultures.
- → Treatment:

PRDS

- 1. Protocol.
- 2. Routes.
- 3. **D**uration.
- 4. **S**tart (when we start?).
- **→** *Prevention:*

↓ risks:

- Caregiver (2).
- Incubator (1).
- Contact (2): (breast feeding & uncover cord).
- Invasive (2): control IV lines & equipment.
- → Supportive & monitoring:
- A. Monitors:
 - 1. Vitals.
 - 2. Gas.
 - 3. CBC.
 - 4. ABG.
- B. Supportive:
 - 1. Temperature.
 - 2. Blood, Acidosis, ...

Cardiology

1. Hypertrophy

 $\mathsf{ASD} \Rightarrow \mathsf{RVH}$

 $VSD \Rightarrow$ bilateral VH but mainly left VH

 $PDA \rightarrow left VH$

2. Treatment of cyanotic spills (central cyanosis)

$$1^{\text{st}} \rightarrow O_2 + IV \text{ fluid}$$
 $2^{\text{nd}} \rightarrow \text{Bicarbonate IV}$
 $3^{\text{rd}} \rightarrow \text{Squatting (knee chest position)}$
 $4^{\text{th}} \rightarrow MV$
 O_2

$$5^{th} \rightarrow \alpha$$
 agonist to + TPR
 $6^{th} \rightarrow \beta$ blocker to relax infundibulum constriction CVS
 $7^{th} \rightarrow Morphine$ – pain

3.

	Fallot's	TGA
<u>Examination</u>		
<u>1-General</u>		
Central cyanosis	1-2 months	At birth
Cyanotic spills	Appear on crying	Appear in 1-2 days
Dyspnea	✓	√ fever
Repeated chest infection	✓	√ fever
Clubbing of fingers	\checkmark	مش هیلحق
Hypoxia test	X	✓
2-Local	Slight pericardial bulge	Huge pericardial bulge (RVH)
Inspection	(RVH)	
Palpation	Systolic thrill over	No thrill
	pulmonary area	But ?? after Rashkind balloon atrial septostomy
Auscultation	 No murmur in (hyper cyanotic spells) Murmur (ejection Harsh systolic murmur) S2 (A2) aortic component 	 No murmur due to heart lesion Murmur after Rashkind balloon atrial septostomy due to mixing blood Single S₂ (A₂) due to anterior replacement of aortic artery

4. 1st line of emergency of central cyanosis at new born is Prostaglandin Infective endocarditis (treatment) medical / ABs ttt 2-4-6 Sugery / Absolute- Relative Rheumatic fever (treatment) (arthritis) = 4 weeks Aspirin (4) (mild Carditis) = 8 weeks (2×4) (sever carditis) = 10 weeks steroid + 6 wks Aspirin (2 x 8) 7. Aortic coarctation treatment: ەن برە >>>graft insertion. stent<<> من جوہ resection >>> 8. Aortic stenosis treatment o من جوه (1st line) Balloon valvoplasty o Valve من ال>>>valvotomy o من بره, valve replacement 9. Wide fixed splitting of P2 P₂ is changed in expiration and inspiration But in case of ASD there will be pleural effusion so lung resistant and so pulmonary pressure will be equal in inspiration and expiration So it's fixed Also splitting ?? wide due to delayed closure of pulmonary valve after aortic valve 10. Murmur of PDA \rightarrow continuous 11. 1st year of life Average BP= 85/50 mmHg 12. Pan systolic = holosystolic murmur. 13. ejection systolic murmur on pulmonary area is: Fallot's tetralogy due to defect in pulmonary stenosis or Atrial septal defect due to increase in blood volume in right ventricle 14. A cyanotic congenital heart present when there is left to right shunt

- 15. Cyanotic disease of heart:
- without shunt → TGA

or

with shunt \rightarrow right to left = TOF

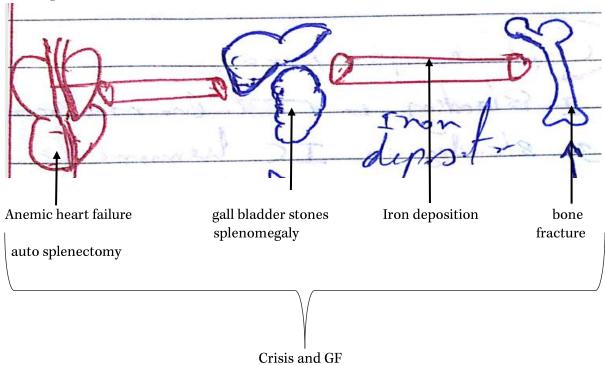
Hematology

- **→** So, C/P:
- General c/p of anemia= loss of function
- ad production \rightarrow red urine, jaundice, \rightarrow deposition \rightarrow wall, core.
- Continuous compensation:
 - Auto compensation
 - Doctor compensation 'Blood transfusion'
- **→** *So, Investigations:*
- Anemia →CBC
- Type: blood film x-ray and blood chemistry
- Causes \rightarrow stool and urine analysis, endoscope.
- Investigation to know type: Blood chemistry (bilirubin indirect) Iron& T1Bc α ferritin.
- **→** *Complications of CHA:*
- 1. Complication of blood transfusion.
- 2. Organomegaly → hepatosplenomegaly & auto splenectomy
- 3. Bad production of iron deposition
 - Core → Bv (cirrhosis) GB (stone)
 - Wall → hemosiderin.
- 4. Loss of function:

Heart failure, growth failure, delayed puberty and easy fracture of the bone.

- → How to prove hemolysis:
- CBC "blood film"
- Reticulocytosis
- Blood chemistry.

2. Complications of CHA



3. Thalassemia انيميا البحر المتوسط

- **→** *Investigations:*
 - CBC
 - Blood film (معض ونصورهم) عشان اعداد کتیره جدا
 - Electrophoresis
 - Genetic study
- **→** *Treatment:*

3S + RBCs

- 1. Supportive
- 2. Splenectomy + Blood transfusion (+Chelating agents)
- 3. Synthesis of Hb
 - Medical: hydroxyurea
 - Surgical:
 - 1- bone marrow transplantation
 - 2- gene therapy
- Supportive ttt → BCDEF

HBV vaccine

<u>C</u>a++

 $\operatorname{vit} \underline{\mathbf{D}}$

Eliminate dietary iron

Folic acid

- Packed RBCs

 $10-15 \text{ ml/kg} \rightarrow 10-12 \text{ ml/dl}$ (MCQ مهمه)

- Iron chelating agents:

deferoxamine

4. Sickle cell anemia

- → Genetics: Gene no 6→7, glutamic by valine
- → C/p of sickle cell anemia or thalassemia

Onset, course, complication + c/p of CHL

- → V.C: ischemia in anatomical parts of the body: LL, UL, head, chest, abdomen and kidney.
- → DD of acute hemolysis (3I):
 - 1- Infection (Hus-relationships).
 - 2- Inherited (G6PD-Wilson).
 - 3- Immune (ABO-AIHA).
- → DD of vasculitis: C 3 I
 - 1- vit c deficiency
 - 2- inherited
 - 3- infection
 - 4- immune

5. HSP

Area	Defects		
Lower limb	100% rash	3/4 joints	
Abdomen	50%GIT	1/3renal	
CNs	Fever	ICH	
Testis	Hemorrhage	Swellings	

6. Leukemia TTT:

- → Induction of remission (AVPC):
- asparaginase
- vincristine
- prednisone
- cytarabine
- → Maintained dose (2M): methotrexate, mercaptopurine.
- → Intrathecal = (SMC) (سمك)
- Cytarabine
- steroid
- methotrexate
- → bone marrow transplantation

7. Hemophilia A:

→ c/p

تحكى قصه حياته من أول الولادة

- Bleeding in circumcision
- Hematoma in minor trauma. بدأ يتعلم المشي وكل شويه يقع
- Hemarthrosis. اول ما مشي رجله ورمت
- Bleeding from orifices.
- Internal hemorrhage "on complication"
- Intramuscular hemorrhage "on complication"

→ Complication:

- Intra Cranial hemorrhage
- Psoas hematoma
- Ankylosis.
- + Complications of ttt.
- → Any investigation in any bleeding disorder
 - CBC
 - bleeding time
 - Clotting time
- detection of clotting factors

→ *TTT*:

- 1. In mild case:
 - cold compression
 - desmopressin
- 2. In severe cases: replacement therapy.
 - Cryoprecipitate
 - purified
- 3. Physiotherapy for all.

8. ITP

- → Clinical picture:
 - 1- Purpura
 - 2- Bleeding in mucous membranes
 - 3- Bleeding in IC haemorrhage

التشخيص thrombocytopenia + antiplatelets Ab <u>لكن مفيش تشخيص بدون</u> <u>bone marrow examination (Very important)</u>

→ *Treatment:*

- Mild cases:
 - 1- follow up
 - 2- avoid trauma and salicylates
- In moderate cases:
 - 1- prednisone
 - 2- IVIG

- In severe cases:
 - 1- prednisone
 - 2- IVIG
 - 3- plasmapheresis
 - 4- platelets + plasma
 - 5- splenectomy
- In chronic cases:
 - 1- prednisone
 - 2- IVIG
 - 3- immunosuppressive therapy
 - 4- splenectomy (75% curative)

chronic: الحل الوحيد في الحالات ال remove your immunity: immunosuppression/ splenectomy

In severe cases (cause of severity) الحل الوحيد انك تشيل السبب cause of damage \rightarrow splenectomy

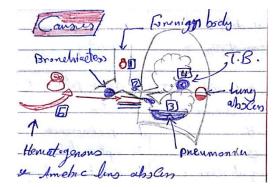
9. Aplastic Anaemia

- **→** *Congenital:*
 - Prolonged survival (androgen and corticosteroids)
 - Bone marrow transplantation is Treatment of choice
- → Acquired:
 - Mild: anti-thymocyte globulin ATG or cyclosporine
 - Severe: BMT or immunosuppression

Respiratory

1. Lung Abscess

- → Causes:
 - 1. Foreign body aspiration
 - 2. Secondary to:
 - a. Bronchiectasis
 - b. Pneumonia
 - c. T.B.
 - d. Metastatic lung disease



- 1) trachea \Rightarrow 2) bronchi \Rightarrow 3)alveoli (foreign body \Rightarrow bronchiectasis \Rightarrow pneumonia)
- 4) parenchyma → Blood vessels (hematogenous + lymphatic)
 - e. Amoebic lung abscess
- → Clinical picture:
 - A. Symptoms:
 - 4. toxemia (pus)
 - 5. cough with sputum + blood
 - B. Signs:

bronchial breathing

→ Complications:

Rupture + fibrosis →suppuration ثَابِتِين فِي أَي (spread + bronchiectasis)

- **→** *Investigations:*
 - Any chest disease → chest X-ray
 - Any abscess → culture and sensitivity
- **→** *Treatment:*

Any abcess:

- AB
- drainage
- surgical correction

2. Pulmonary Tuberculosis

Spread to spaces

Alveoli

Spread to bronchi Spread in blood vessels

→ Clinical picture:

- 1^{ry} focus:

no symptoms or signs

- 2ry focus: (active 1ry focus):
 - A. Symptoms:
 - 1. T.B. toxemia
 - 2. cough + sputum
 - 3. history of false diagnosis as pneumonia

B. Signs:

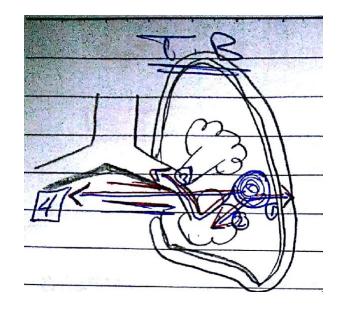
- 1. pleural effusion
- 2. pneumonia
- 3. fibrosis
- 4. compression

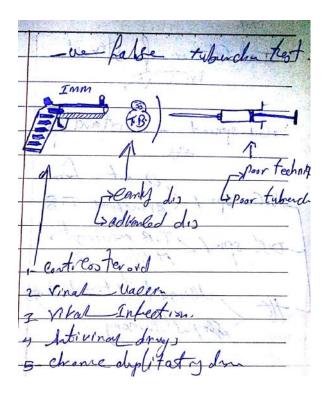
→ false -ve tuberculin test

- 1- early disease
- 2- advanced disease
- 3- poor technique
- 4- poor tuberculin
- 5- corticosteroids
- 6- viral infection
- 7- antiviral drugs
- 8- chronic debilitating disease

→ *Isolation of organisms through gastric aspirate:*

- 1. ZN stain
- 2. Culture \rightarrow Lowenstein Jensen \rightarrow BACTEC
- 3. Test \rightarrow QuantiFERON TB ELISA PCR





- → *Indication of steroid in TB*:
 - 1- Local spread
 - Exudative form
 - Bronchial TB
 - 2- Distal spread:
 - TB meningitis
 - Military TB
 - 3- Treatment:
- Allergy to anti-tuberculous drugs
- Post-surgery
- Commonest organism in common cold (rhinitis) → rhinovirus
- Commonest organism in pneumonia → pneumococci
- Most cases of bronchitis resolve spontaneously
- Bronchodilators are not effective in bronchiolitis
- Bronchitis → dry cough → then → productive cough → then → dry cough
- Pleural effusion & lung abscess present as a complication only in case of staph pneumonia
- Finger clubbing associated with suppurative lung disease
- Tuberculin test appears after 2-3 days (24-72 hrs.)
- INH → Drug of choice in TB & causes → jaundice
- IgE is not essential for diagnosis of bronchial asthma as there are 2 types IgE related BA/non-IgE related BA
- Respiratory syncytial virus → intracellular (interstitial pneumonia) → wall of bronchioles → constrictions → wheezes
- wheezes → may be in laryngitis if the cause is RSV
- Life threatening conditions = causes of stridor + causes of RF
- Extrinsic asthma = pollens, mites, house dust/ hereditary/ IgE
- intrinsic asthma = infection, smoke, emotional, viral, exercise.
- Tonsillectomy indications:
 - 1- chronic tonsillitis
 - 2- peritonsillar abscess
 - 3- tonsillar abscess
 - 4- recurrent acute tonsillitis more than 4 times per one year
 - Atypical pneumonia = mycoplasma & TB pneumonia
 - Peak age of acute bronchitis → 6 months of age
 - Bronchiectasis predisposing factors are chronic causes
 - Commonest cause of wheezes in: children → bronchial asthma infancy → bronchiolitis

3. Suppurative Lung Disease

	Lung abscess	Bronchiectasis	Emphysema	
Definition	Suppurative destruction of lung parenchyma → cavity+ pus	Bronchial dilatation→ pus stagnation	Pus in pleural cavity	
Causes	 Aspiration of foreign body 2ry to pneumonia bronchiectasis TB amoebic lung abscess metastatic lung abscess 	- Congenital malformation - Acquired chronic infection: 1- F.B. 2- T.B. 3- lung abscess 4- immotile cilia syndrome 5- cystic fibrosis 6-GERD	Sterile potential space 2ry to: 1- pneumonia 2- rupture of lung abscess 3- chest trauma/surgery 4- mediastinitis 5- sub diaphragmatic abscess	
Symptoms	clubbing of fingers	l , anorexia, weight loss, opious, purulent, foul ural variation and	- FHMA - Chest pain(dyne): ↑ cough ↓ lie on the affected side	
Signs	- Inspection: Palpation: Auscultation→ localized bronchial breathing	 Inspection: Palpation: Auscultation: patchy bronchial breathing + coarse crepitation near the affected region 	 Diminished air entry Dullness Diminished breath sounds 	
Complications	Rupture → spreadfibrosis→bronchiectasis	rupture →growth failurefibrosis → RF	rupture → fistulafibrosis →chronicity	
Investigations	X-raysputum culturebronchoscopy	- X-ray - CT	X-raypus culturethoracentesis	
Treatment	 - AB 4-6 ms - Drainage (physiotherapy) - Surgical correction - Bronchoscopy in case of F.B. 	 AB 4-6 ms Drainage (physiotherapy) Surgical correction Bronchodilators 	 AB 4-6 ms Drainage chest tube Surgical correction Modified AB according to culture 	

4. Cystic Fibrosis

	Pancreatic	Biliary	Respiratory	Immotile cilia syndrome
Symptoms in infancy	Non-digested meconium → accumulation → meconium ileus→	Prolonged neonatal jaundice irritation to	Accumulation of sputum → recurrent chest infection	
	accumulation→ rectal prolapse	hepatocytes		
Children	Non-digestion → non- absorption→ steatorrhea → failure to thrive	chronic hepatitis	chronic → bronchiectasis/ emphysema rupture	Nasal polyp Sinusitis
Adolescents	No digestion → no absorption → no insulin secretion (DM)	cirrhosis + portal HTN	pneumothorax or recurrent haemoptysis	Sterility in male

GIT

- Any congenital abnormality in the GIT appears before 1 month.
- Neuroblastoma < 3 years.
- Wilms` tumor (Nephroblastoma) ≥ 3 years.
- Vomiting \rightarrow alkalosis.
- Diarrhea → acidosis.
- ORS 150-300 ml/kg/day.
- hypokalemia → abdominal distention paralytic ileus-
- convulsions → sweating hypernatremia dehydration
- ORS in all diarrhea & dehydration except shock
- Parvovirus \rightarrow commonest virus in respiratory infections.
- Retrovirus \rightarrow commonest virus in GIT infections.
- Vomiting after period of 2 months \rightarrow CHPS.
- Diarrhoea at period of weaning \rightarrow celiac disease.
- GRD during 1st year of life.
- Low grade fever in GE \rightarrow Rotavirus.
- DKA → acute abdominal pain
- Source of infection of Monilial stomatitis [Nipples, Perineum] of the mother
- We don't give Anti-mobility drugs in diarrhea if gastrointestinal infection exists: -
 - 1- Will increase bacterial stasis.
 - 2- Will increase toxin absorption.
 - 3- Will increase epithelial damage.
- $RE \rightarrow Bleeding \rightarrow intussusception.$
- الشبسي كله املاح = بيبوظ املاح الجسم CHPS
 - So, electrolyte imbalance so we do serum chemistry
 - & so, we should correct blood chemistry before surgery not emergency surgery
- **CHPS**

Four Fs

- Male to \underline{f} emale = 4/1
- more in first born boy
- positive <u>f</u>amily history
- multi factorial

سىنارىو

-CHPS-

الام هتقولك : الولد جعان على طول أول ما يأكل يرجع و جسمه خاسس الدكتور حضرتك هتعمل

 \rightarrow *Test feeding* \rightarrow visible peristalsis

Olive-like mass

- → Investigations: احنا قلنا الشيبسي كله املاح هيبوظ كيمياء الدم
 - U/S Barium meal
 - Serum chemistry
- → TTT: 1st correct serum chemistry

- Small GIT obstruction \rightarrow Volvulus.
- Lower GIT obstruction \rightarrow Intussusception

Hepatology

1. Portal HTN > 12mmhg	
Causes	- prehepatic
	- hepatic {sinusoidal}
	- post hepatic
c/p	Collaterals. bleeding, Ascites, splenomegaly.
	- Ascites: post hepatic or post sinusoidal to occlude the flow (tall
	columnar)
	- Splenomegaly: prehepatic or hepatic
	- Prehepatic or hepatic → still low pressure so fragile organs can compensate
	- Post sinusoidal or post hepatic → high pressure
	So larger surface area & organ can compensate
Investigations	Vascular disease due to hepatic cause
	- endoscopy → VARICOSE
	- CT, MRV
	- DOPPLER & US
	+
	HEPATIC causes
	1- Liver function ALT, AST, CBA
	2- MARKERS& TORSH SCAN
	3- TMS
	4- autoimmune enzymes
	5- liver biopsy
Management	Bleeding →IV fluid
	→vitK, plasma, blood
	Endoscopy→ vasopressor infusion
	Sclerotherapy & band ligation
	Surgery →TIPSS
	→PSS
	PREVENTION OF BLEEDING
	1 ST attack
	1- Avoid ASPIRIN &NSAI
	2- BB
	3- Protective endoscopy
	Re-bleeding

1-BB
2-PROTICTIVE ENDOSCOPY
3-TIPSS
4-PSS
5-LIVER TRANSPLANT

2. CHOLESTASIS	
Causes	- Hepatocellular
	1- Idiopathic
	2- Inflammatory
	3- Metabolic storage
	- Intrahepatic duct → familial Cholestasis
	- Extra hepatic duct → EHBA &
	choledochal cyst.
C/P	- Weight →NORMAL in EHBA
	→decrease in congenital and inflammatory
	- Specific to the cause
Investigations	- LIVER function
	- Prove cholestasis
	- Prove the causes and complications
	- 5 steps diagnosis
	1- INV treatable conditions
	2- TORSH screening
	3- OTHER METABOLIC conditions
	4- U/S
	5- Differentiate BETWEEN
	CHOLLEDOCAL CYST & HEPATITIS by
	LIVER BIOPSY and
	HID Scan
TTT	- Replacement therapy
	- Displacement surgery
	- Symptomatic
	1- Pruritic
	2- Varicose
	3- Encephalopathy
	4- Growth failure
	- Liver transplant

3. Hepatology classification

- 2 infections (acute or Chronic) = {ABCDEF}
- 2 obstructions
- طالع: PORTAL HTN CAUSES {PREHEPATIC, HEPATIC, POSTHEPATIC}
- نازل : Cholestasis: causes {hepatocellular, intrahepatic obstruction, extrahepatic obstruction}

4. Cirrhosis

- 1- chronic hepatitis {liver}
- 2- Cholestasis نازل hepatocellular and ductal
- 3- Congestive طالع heart failure

5. Acute liver failure

Functional liver failure

1- SYNTHESIS

there will be deficiency in:

- Albumin
- Clotting factors
- Billirubin
- 2- DETOXIFICATION

there will be increase in blood level of:

- Drugs
- Urea
- Toxin
- 3- REMOVAL

there will be increase in blood level of:

- CHOLST
- Bilirubin
- BILE Secretion

Neurology

1. Acute muscle weakness

Guillain barre syndrome

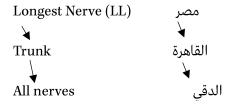
→ Definition:

GIT infection \rightarrow auto Abs attached to myelin sheath \rightarrow polyneuritis \rightarrow acute loss of motor function.

→ *Incidence:*

GIT infection \rightarrow GBS 50% of child disease (Most common cause of acute paralysis of children).

GBS → GPS (Maps)



- **→** Examination:
- Motor → Ascending symmetrical
- Sensory
- Autoimmune
- **→** *Investigation:*

Diagnosis (clinical)+General look

- → *Management:* (respiratory-CVS- CNS)
- Respiratory (ICU- Mechanical ventilation)
- CVS (IVIG- plasmapheresis)
- CNS: physiotherapy
- → Focus on:
 - 1) Acute LMNL
 - 2) Paradoxical breathes
 - 3) Bulbar paralysis
 - 4) Symmetrical weakness.

2. Progressive muscle weakness

I. Duchenne muscular dystrophy

- → Most common & most serious muscular dystrophy
- → *Diagnosis:* CPK- Gower signs

Search for mother family history (X linked recessive disorder)

- **→** C/P:
 - 1. Hypotonia (gridle (pelvic-shoulder)- trunk)
 - 2. Cardiomegaly
 - 3. Pseudo hypertrophy

- 4. Mental affection
- 5. Skeletal deformities

II. Floppy infant

- → Clinical:
 - Limbs: frog legs
 - Trunk: curved
 - Head: lag

III. Werdnig Hoffman

- Worm like movement
- Can't sit or walk (But grade II can sit) and (Grade III: delayed walk paralysis)
- **→** *c/p*:

disease of spinal cord so:

- normal mentality
- normal eye movement
 - Prenatal:
 - Diminished fetal movement
 - arthrogryposis
 - Perinatal
 - floppy infant
 - decreased tendon reflex
 - Postnatal
 - bulbar palsy
 - tongue fasciculation
 - Late
 - respiratory paralysis & RF
- **→** *Investigations:*

Muscle:

- EEG
- biopsy
- molecular study

3. MR

- → Causes:
 - A. Hereditary

1. Chromosomal (number- structure)

بالوراثة

- 2. Genetic (microcephaly)
- 3. Error of metabolism
- 4. Neurodegeneration
- 5. Neuro-cutaneous
- B. Acquired (prenatal natal-postnatal)
- → Management
 - 1. Prevention
 - 2. Mother (at home) (specific ttt-general)

- 3. Physiotherapy- Ortho therapy- speech- ophthalmology
- 4. Antiepileptic + educable child.

4. CP:

- → Causes:
 - 1. Congenital
 - a. Malformation
 - b. infection
 - 2. Acquired (prenatal natal- postnatal) as MR.
- **→** C/P:

Vague but suspected by

- 1. Mother (feeding-gagging)
- 2. Paediatrician (microcephaly- development)
- 3. Neurologist (abnormal limb tone- persistent neonatal reflexes)
- **→** *Type of palsy:*
 - Spastic (متخشب) (hypertonic- hyperreflexia)
 - Atonic (مرخخ بس قوى الرفلكس)
 - Diakinetic (زى الدودة)
 - Ataxic (مرخخ و بیترعش و بیطوح)
- → Complications (*muscle chest- association*)
- **→** *Investigation*:
 - TORCH
 - CT
 - VEP
- **→** *Management:*
 - conservative (حاول تصلب جسمه)
 - Treat Complications.

5. Meningitis

→ *Complications:*

Early → spread

Late \rightarrow localization fibrosis

- Toxins (sensory & motor defect/ motor irritation epilepsy)
- Fibrosis (hydrocephalus)
- Mass Abscess pressure (empyema-ICP)
- **→** *Treatment:*

Start by empirical therapy

	Duration	Ttt
Before 2 months	2 -3 w: neonate	Ampicillin
	7 – 10 days: 2 nd month	Cefotaxime
Above 2 months	7 -10 days	Cefotaxime
		May be Chloramphenicol

⁺ steroid in H. Influenza (↓ hearing loss)

→ Notes:

- Most common organism
 - GBS (Bacteria)
 - Enterovirus (virus)
- irritation signs: absent in infant 2- 18 months
- Chorea: involuntary movement involves proximal muscles which disappear during sleeping.
- H. Influenza meningitis (the only) \rightarrow non-pyogenic meningitis \rightarrow are given steroids.
- GBS, most common organism in neonatal meningitis

6. Drugs used as DOC (Drug of Choice):

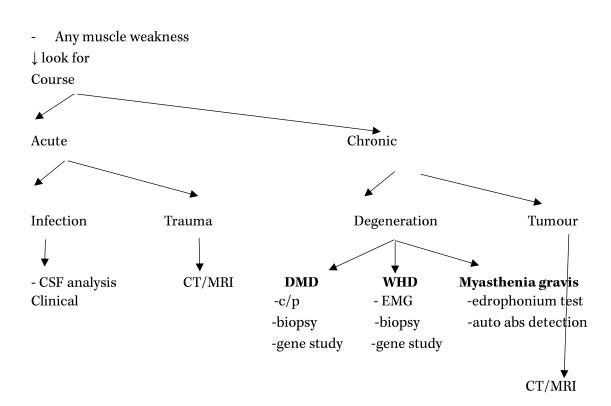
- Resistant seizure: lamotrigine
- generalized absence: Na valproate
- absence: Ethosuximide
- myoclonic: Clonazepam
- Tonic-colonic seizure at the age 5-15 years
- Neonatal seizures are myoclonic & infantile spasm

Most common:

- 1. Cause of hydrocephalus: Aqueduct stenosis.
- 2. Cause of floppy infant: werdnig Hoffman
- 3. Cause of acute muscle paralysis: GBS
- 4. Cause of muscular dystrophy: DMD
- 5. Cause of bacterial meningitis →: GBS
- 6. Cause of viral meningitis: enterovirus

General notes:

- EEG in seizure
- EMG in GBS, DMD, WHD
- DMD & WHD we need muscular biopsy & genetic study
- Neonatal seizure prognosis (poor hypoglycaemia -hypocalcaemia)
- Typical febrile convulsion (normal EEG- generalized in nature)



Endocrine

1. DM

	Type l	Type 2
Incidence	90%	10%
<u>Causes</u> - genetic	Polygenes	Polygenes
-autoimmune	Yes	No
<u>Defect</u> -insulin secretion	-	Variable
-Insulin receptors	No resistance	Resistant
<u>Result</u> ketosis	Common	Rare
<u>Ttt</u> insulin dependence	Total	Uncommon

→ Pathophysiology

Insulin (- glucose)

Insulin antagonist (+ glucose)

- growth hormone
- Glucagon
- Cortisol

There is balance between insulin and its antagonist So if there decrease in insulin ... insulin antagonist will work on many organs as

- 1- Liver → gluconeogenesis
- 2- Fat → ketosis
- 3- Muscle → gluconeogenesis

- ightharpoonup c/p: ماء ightharpoonup polyuria, enuresis خار fever DKA
- **→** *Complications:*
 - 1. Acute
 - hyperglycemia→DKA, infections
 - hypoglycemia → من العلاج الزيادة

2. Chronic:

Glucose bind to BM of BV

So micro vasculopathy

- Retina
- coronary artery {IHD}
- nerves

Plus

- A/autoimmune thyroiditis
- P/puberty delay
- C/celiac disease
- **→** *Investigations:*
 - DM
 - DKA
- **→** *DD*:
 - Of DKA: Coma/Metabolic acidosis/Acute abdomen/Vomiting/Dehydration/RD.
 - Of DM: Polyuria/2ry nocturnal enuresis/Failure to thrive/Immunodeficiency.
- \rightarrow TTT of DM:
 - 1- Hospitalization
 - 2- Insulin =DINRR, T / monitoring glucose
 - 3- Diet
 - 4- exercise

2. Puberty

→ Manifestations: سهلة ومهمة

1ST sign in

- Male: testicular enlargement
- Female: breast enlargement

Last sign in:

- Male: height spurt
- Female: menstruation (and gaining 5 cm height ملهاش لزمه. قولها في الشفوى بس

SMR (sexual maturity rating) in female: {breast, pubic hair}

SMR in male: {pubic hair, testis & penis}

- I. Delayed puberty
- 1- Constitutional
- 2- Low gonadotropin

Pituitary {systemic disease, starvation, hypothalamo-pituitary disease}

- 3- Testes {high gonadotropin}:
 - a. anatomical = gonads damage
 - b. functional {chromosomal, enzymes}

II. Precocious puberty

- A. Only one character
 - 1- premature thelarche, adrenarche
 - 2- gynecomastia in male
- B. All 2ry sexual characters
 - 1- true {with gametogenesis} increases gonadotropin
 - 2- false {without gametogenesis} increased estrogen or androgen

3. 1ry Hypothyroidism.

- → Causes:
 - A. Congenital:
 - 1- anatomical defect {maldevelopment}
 - 2- functional defect
 - a- dyshormonogenesis
 - b- iodine deficiency
 - c- goitrogens
 - B. Acquired:

Hashimoto's

- **→** *c/p:*
 - at birth: no symptoms but screening
 - early:
 - 1- increase in gestational period
 - 2- symptoms الولد بينام لحد ما اصفر من قلة الاكل
 - 3- Signs راسه وایده وجلده وبطنه (APCD)
 - LATE: شيت الاطفال
 - A- HISTORY → development
 - **B-** Examination
 - 1- Measures
 - 2- H&N
 - 3- Limbs
 - 4- Skin
- **→** *Investigations:*
 - 1- iodine assay
 - 2- Thyroid u/s
 - 3- Laboratory finding $\{T_3, T_4\}$
 - 4- bone age x-ray
- **→** *TTT*:
 - 1- LIFE LONG THERAPY
 - 2- MONOTRING
 - a- Develop
 - 1- Motor
 - 2- Mental
 - 3- Puberty